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# EPILEPSY

AND OTHER

# CHRONIC CONVULSIVE DISEASES:

*THEIR CAUSES, SYMPTOMS, & TREATMENT.*

BY

W. R. GOWERS, M.D., F.R.C.P.

ASSISTANT PROFESSOR OF CLINICAL MEDICINE IN UNIVERSITY COLLEGE;
SENIOR ASSISTANT PHYSICIAN TO UNIVERSITY COLLEGE HOSPITAL;
PHYSICIAN TO THE NATIONAL HOSPITAL FOR THE PARALYSED AND EPILEPTIC.

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J. & A. CHURCHILL, NEW BURLINGTON STREET.
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151. q. 45.

DEDICATED

TO

J. RUSSELL REYNOLDS, M.D., F.R.S.

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS
PHYSICIAN TO HER MAJESTY'S HOUSEHOLD
CONSULTING PHYSICIAN TO UNIVERSITY COLLEGE HOSPITAL

WHOSE EXAMPLE HAS STIMULATED
AND FRIENDSHIP ENCOURAGED
THE WORK CONTAINED IN THE FOLLOWING PAGES

# PREFACE.

THE object of the following work is to describe and illustrate the clinical history of Epilepsy and other allied convulsive diseases by the help of the material furnished by a series of cases which have been under my care, chiefly at the National Hospital for the Paralysed and Epileptic. Some of the facts of these cases, and the conclusions which they suggest, were described in the Gulstonian Lectures delivered before the Royal College of Physicians of London in February 1880. Those facts and conclusions are here reproduced, in their proper place, in the systematic account of these diseases. The lectures, as published in the medical journals, do not, however, occupy more than a fourth part of the present volume.

Many of the conclusions given in the following pages are the same as those which other workers have reached; others are different. It has not been practicable to refer, in every instance, to the numerous writers who have expressed opinions on the several debated points. Whatever value is possessed by the inferences here presented is due to their being drawn from an extensive series of facts in an original investigation. I have, however, referred to the statements and opinions of others whenever justice or the importance of the facts seemed to render such reference desirable.

QUEEN ANNE STREET:
*September* 1881.

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# EPILEPSY

## AND OTHER CONVULSIVE DISEASES.

### INTRODUCTION.

THE convulsions which occur as a result of chronic brain disease may be divided into two classes: (1) Those which are the result of organic disease, such as can be recognised, after death. (2) Those which are the expression of a condition of the brain which is not evidenced by any visible alteration. The only evidence of disease being the disordered function, they are termed 'functional' diseases.

The object of this work is to consider the clinical history of the chronic convulsive diseases of the latter class; which depend on such conditions of the nerve centres as elude detection by the methods of examination at present at our disposal. In considering these diseases it will, however, be necessary to refer to many facts relating to the organic diseases of the brain. There is, moreover, one class of convulsions due to organic brain disease which it has seemed well to consider more fully, namely, those which succeed an attack of hemiplegia from which a more or less complete recovery has taken place.

The chronic convulsions which are recognised as of 'functional' origin are commonly divided into two classes —the epileptic and hysterical attacks. The former are characterised by almost invariable loss of consciousness and special forms of spasm, tonic or continuous, clonic or intermitting. In severe attacks (epilepsia major, *grand mal*) both forms of spasm usually occur. The muscular contractions are of violent random character, fixing the limbs in unnatural attitudes, unlike those which are assumed during voluntary movements. In slighter attacks (epilepsia minor, *petit mal*) there is transient loss of consciousness with little or no convulsion. In some rare attacks (less common than in cases of actual organic disease) there is slight convulsion without loss of consciousness.

The attacks which are commonly called 'hysterical' vary much in their characters. There may be merely trifling emotional and spasmodic disturbance, such as is commonly understood by the designation, or there may be most severe and long-continued spasm, apparently rivalling a severe epileptic fit in the violence of the muscular contractions, attended with impairment, if not actual loss, of consciousness, and often with paroxysms of delirium. But the chief part of the muscular spasm which occurs in these attacks differs from that of an epileptic fit in being so grouped as to resemble that which may be produced by the will. The convulsive movements have therefore a quasi-purposive aspect, they are *co-ordinated* in character, although excessive in degree (rigidity, opisthotonos, throwing about of limbs, bounding movements, &c.) At the onset there may be tonic or clonic spasm (a pseudo-epileptic stage), but this rarely resembles closely that which occurs in epilepsy. These severe fits often suggest the idea of demoniacal possession rather than of hysteria, and whatever abstract truth there is in applying to them the designation 'hysterical,' the trivial meaning commonly attached to the term renders it a misleading name. Dr. W. Roberts has proposed to call such attacks 'hysteroid;' and this term, to whatever etymological objection it may be open, is so convenient that I have commonly employed it. The character of the attacks may also be accurately described by the term 'co-ordinated convulsion,' which I have long used and found of great convenience when it is desirable to avoid reference to the underlying state. These severe hysteroid fits have long been known also under some hybrid term, as 'epileptic hysteria,' 'hysterical epilepsy,' or 'hystero-epilepsy,' and to the last designation the writings of Charcot have given wide currency. But the term tends rather to hinder than advance the study of the nature of these convulsive attacks, and their relation to other forms of seizure, and it seems to me a clear advantage to discard its use as far as possible.

The separation of these severe hysteroid convulsions from cases of simple epilepsy is less easy than might be inferred from the difference above described between the two classes of seizures, and from the sharp distinction currently drawn between them. The severe hysteroid fits may recur, during years, in very much the same manner as do epileptic fits, and many of these cases have, I am convinced, been included in all published statistics of epilepsy. Moreover, hysteroid or co-ordinated convulsion often succeeds a true epileptic fit; *i.e.* many hysteroid fits are really post-epileptic phenomena. It is often most difficult, even impossible, to learn, from the description of hysteroid convulsions, whether they occur alone, or whether they succeed slight epileptic seizures. The difficulty is increased by the fact that the initial convulsion of many pure hysteroid fits is pseudo-epileptic, *i.e.* bears some resemblance to that of epilepsy. I believe further, and shall endeavour to show, that there are rare cases in which the attacks are actually of a nature intermediate between the two.

Thus the two forms of disease closely intertwine, and to separate altogether the cases of chronic convulsive disease with, from those without, hysteroid symptoms, would involve a large amount of error. A number of the cases which would be placed in the latter group, the hysteroid, really belong to the former, the epileptic, and how large the proportion is could not even approximately be determined.

In the attempt made in the following pages to ascertain, by numerical analysis, some of the facts in the history of these diseases, I have not, therefore, attempted an entire separation between the two classes. I have, it is true, rejected those cases in which patients, presenting other indications of hysteria, had only slight hysterical fits, without marked loss or perversion of consciousness, but I have included in the series analysed those cases in which, although the visible convulsion was of hysteroid type, the general history of the disease, in its long duration, and the recurring character of the attacks, resembled that of epilepsy. But while it is not possible, for the reasons given, to separate entirely the two groups, it seems desirable to ascertain the extent to which the conclusions reached apply to each group (as far as they can be distinguished), as well as to the whole series. I have therefore, on most points, ascertained the facts, first with regard to the whole series, and then with regard to each group, (1) those with pure epileptic attacks, and (2) those with hysteroid (coordinated, or hystero-epileptic) seizures. By distinguishing the results obtained with reference to the two classes, some important etiological and diagnostic facts are brought out. The subject of the relation of these two forms will be further discussed in the chapter on Pathology.

The cases from which the facts have been ascertained are an unselected series which have been under my care, chiefly at the National Hospital for the Paralysed and Epileptic. The total number of cases is 1,450; but all are not available for comparison upon every point. In each instance the facts have been ascertained from as large a number as possible, and the number is stated in all cases.

Before going further, it may be well to ask what is the relative proportion of the cases presenting these two classes of symptoms, the epileptic and the hysteroid. In determining this point it was necessary to exclude a considerable number of cases in which the facts, which could be ascertained regarding the attacks, were not sufficient to decide their nature. In 1,000 cases, however, in which an opinion, reasonably exact, could be formed, the attacks were purely epileptic in 815, while hysteroid attacks occurred in 185, or 18½ per cent. This does not profess to represent the actual proportion of pure hysteria to epilepsy, but it probably represents, with fair accuracy, the proportion of the cases with recurring convulsions of epileptic and of hysteroid type.

# CHAPTER I.

## *ETIOLOGY.*

THE causes of epilepsy may be divided into those which are remote, and those which are immediate. We must regard as the immediate or exciting cause that to which the first fit was apparently due. Of the two classes of causes, the former, the remote, is incomparably the more important. Most of the immediate causes of the first fit, although apparently influential in exciting it, are manifestly inadequate, alone, to account for its occurrence. Their effect is rendered possible by the antecedent predisposition. In a large number of cases no exciting cause can be ascertained; the first attack is then apparently the result of the predisposition only.

When one attack has occurred, whether in apparent consequence of an immediate excitant or not, others usually follow without any immediate traceable cause. The effect of a convulsion on the nerve centres is such as to render the occurrence of another more easy, to intensify the predisposition that already exists. Thus every fit may be said to be, in part, the result of those which have preceded it, the cause of those which follow it. The search for the causes of epilepsy must thus be chiefly an investigation into the conditions which precede the occurrence of the first fit.

### PREDISPOSING CAUSES.

*Sex.*—The first etiological condition to be considered is the influence of sex. Females suffer from chronic convulsions rather more frequently than males. In the total number of cases analysed, 1,450, females preponderate over males, nearly in the proportion of 6 to 5. More exactly, the percentage of females is 53·4; of males 46·6; that is, to every hundred males there were 114 females. This is not due to accidental circumstances, for at the hospital in which most of these observations were made, in the cases which do not present convulsion, males preponderate. It may be thought that this excess of females is due to the inclusion of hysteroid cases. But this is true only to a small extent. In the cases of pure epilepsy the females are still in excess, amounting to 52 per cent., the males constituting 48 per cent. In the cases presenting hysteroid symptoms, females preponderate to a much larger extent, and are to the males as 2 to 1, 66 per cent. being females, 33 per cent. males.

In most statistics of epilepsy hitherto collected in this country males have preponderated. This is probably due to the inclusion of cases of syphilitic brain disease, which we have only lately learned to separate. The proportional affection of the two sexes given above is nearly the same as that ascertained by Herpin.[1]

*Hereditary Predisposition.*—There are few diseases in the production of which inheritance has greater influence. In examining its effect, it is necessary to enquire (1) how is its existence manifested? (2) in what proportion of cases can it be traced? (3) how far does it influence (*a*) the form of the resulting disease, and (*b*) the age at which the affection commences?

It is well known that the neuropathic tendency does not always manifest itself in the same form. On the other hand, we cannot regard all forms of disease of the nervous system as evidence of its existence. Many diseases, for instance, are the result of morbid states of the blood-vessels, and even of organs, as the heart and kidneys, which are far removed from the nervous system. Thus we cannot regard apoplexy and hemiplegia as related to epilepsy.[2] The chief other morbid states, (besides epilepsy) in which the neuropathic tendency is manifested, are insanity, and, to a much smaller degree, chorea, hysteria, and some forms of disease of the spinal cord. Intemperance is probably also due, in many cases, to a neuropathic disposition, but is so common among the poor that its existence can hardly be taken as evidence of disease.

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[1] *Du Prognostic et du Traitement de l'Epilepsie.* Paris, 1852.

[2] Herpin, who carefully investigated this question, found that the fre-

In all investigations into inherited tendencies the facts ascertained fall short of the truth on account of the conditions of the investigation,—disease among the relations, although existing, not being ascertainable. This source of fallacy has probably less influence in the case of the disease under consideration than in that of any other form of disease. The existence of fits or insanity in any individual is generally well known throughout the family, and is not readily forgotten. Moreover, among the poor, there is less tendency to conceal such facts than there is among those in better social circumstances.

The great bulk of the facts collected relate to epilepsy and insanity. Of hysteria little evidence could be ascertained, and in very few cases was there a history of forms of paralysis which could be considered as related in their nature. No doubt, by this method, the facts obtained scarcely adequately represent the actual heredity, but I believe they comprehend all that is with certainty ascertainable, and in such an enquiry it seems better to be within the truth than beyond it.

The proportion of cases in which evidence of inheritance is to be traced was found by Echeverria [1] in 300 cases to be 28 per cent., by Reynolds,[2] from a smaller number, to be 31 per cent. In my own cases the proportion is rather higher. Of 1,218 in which the point was carefully investigated, 429, or 35 per cent., presented evidence of neurotic inheritance. There is no difference in the frequency of

---

quency of apoplexy among the relations of epileptics was positively smaller than in the population at large.

[1] *On Epilepsy.* New York, 1870, p. 183.

[2] *Epilepsy,* &c. London, 1861, p. 124.

inheritance in the two varieties: in each it is nearly 36 per cent.—that is, roughly speaking, of every eight cases of epilepsy, or of severe hysteroid fits, on an average three will present evidence of neurotic heredity and five will not. Inheritance does not affect the two sexes equally, as is shown by the following figures:—

| | Males | Females | Total |
|---|---|---|---|
| Without heredity . . . | 390 | 399 | 789 |
| With heredity . . . . | 194 | 235 | 429 |
| Total . | 584 | 634 | 1218 |

In the cases in which there was no inheritance, the males amounted to 49·4 and the females to 50·6 per cent., but among those in which the disease was inherited the females constituted 55 and the males only 45 per cent. It seems, therefore, that when there is an inherited taint the females of a family are more likely to suffer than the males. The same fact has been noted by other observers. In examining the mode of inheritance we obtain some light on the origin of this excess.

The inheritance is from the mother's side rather more frequently than from the father's, the difference amounting to 6 per cent., as shown in the following table:—

| Inheritance | Cases | Percentage |
|---|---|---|
| Father's side . . . . | 146 | 34 |
| Mother's side . . . . | 169 | 40 |
| Both sides . . . . . | 25 | 5 |
| Collaterals only . . . | 89 | 21 |
| | 429 | 100 |

The side from which the disease is inherited is not unimportant, for it has a distinct influence on the occurrence of the affection in the two sexes. There is clearly a tendency for the transmission to be from father to son and from mother to daughter. In the cases in which the disease was inherited from the father's side, males were in excess of the females by about 4 per cent. (76 males to 70 females, equivalent to 52 per cent. males and 48 per cent. females), which is an excess of 6 per cent. above the average for the whole number (46 per cent. males).

Whereas, in the cases in which the disease was inherited from the mother's side, females were in excess by about 16 per cent. (98 to 71). Thus the percentage of the males affected is 20 per cent. greater when the disease is inherited from the father's than when from the mother's side.

The affection of brothers or sisters must be regarded as an indication of an inherited taint, although we may be unable to trace antecedent instances. In these cases, 89 in number, the females are in very large excess—53 females to 36 males; but the total number is probably too small to permit safe generalisation.

We may now see, in part at least, why females preponderate so much more in the cases of inherited disease than when there is no inheritance. First, the inheritance is more frequently from the mother's side than from the father's; and, secondly, when the inheritance is from the mother's side, girls suffer in a much larger proportion than boys.

Of the relatives who suffer, the mother is herself diseased in a much smaller proportion of cases than the father. The father himself was the subject of neurotic disease in 45 per cent. of the cases in which the inheritance was paternal; the mother, in only 35 per cent. of the cases in which the inheritance was maternal.

Of the neurotic diseases which can be traced in the antecedents of epileptics, epilepsy is incomparably the most frequent. A history of epilepsy was obtained in nearly three-quarters of the inherited cases. In rather more than half (240) it existed alone; in 54 it was combined with insanity, in 5 cases with chorea, and in 3 with both insanity and chorea. Insanity occurred in the relatives of about one-third (157) of the inherited cases, and of these it existed alone in two-thirds. Chorea occurred in other relatives in 35 cases, in 24 of which it existed alone; in 5 it was associated with epilepsy, and in 3 with insanity. The forms of paralysis, paraplegia and infantile palsy, included in the investigation, were not more frequent in the families with other evidence of neurotic tendency than in those who presented no such history. The cases in which an antecedent family history of hysteria was obtained were very few.

Although the mother was herself less frequently affected than the father, the two suffered from epilepsy with equal frequency, and the difference in the frequency of their affection depends chiefly upon the fact that the mother suffered from insanity much less frequently than the father. Of 394 cases of inherited epilepsy, the father was epileptic in 45, and the mother in 46 cases; the father was insane in 21 cases, the mother in only 11.

In some cases the family tendency, as evidenced by the number affected, was very strong. In one case, for instance, five other individuals were epileptic—the patient's mother, maternal aunt, two uncles and cousin. In another instance no less than fourteen members of the family suffered from epilepsy—the patient's mother and maternal grandmother; her mother's sister and mother's brother's son; four of her own sisters, and five of her sisters' children.

*Heterogeneous Heredity.*—Is epilepsy predisposed to by any other inherited tendency than that indicated by the occurrence of its own allies? Rheumatism and phthisis have both been supposed to favour its occurrence. Regarding *rheumatism*, apart from the heart disease which it may cause, I have been able to obtain no evidence in support of the view. The effect of *phthisis* is very difficult to ascertain. It is certain that the family history of epileptics presents a large proportion of cases of phthisis, but in the opinion of some writers, as Nothnagel, the frequent coincidence is accounted for by the commonness of lung disease. Others, as Echeverria, believe that the frequent association supports the view that there is a causal relation between the two diseases, and Savage has expressed a similar opinion with regard to insanity. That there is a relationship between the two is suggested strongly by individual cases, as, for instance, that of a patient, who, presenting no evidence of neurotic inheritance, had lost father, mother, and six brothers and sisters from phthisis, she herself, the sole survivor, being epileptic. The statistics which I can offer on this point are unfortunately not very extensive; as far as they go they do not give support to the opinion that there is a relationship between the two diseases. Phthisis was enquired for in 300 cases, and a history of it in parents, grandparents, brothers or sisters, uncles or aunts was obtained in 108, or 39 per cent. Considering the commonness of phthisis, this does not seem a larger proportion than might have been expected, although I am not aware that there are any statistics which can be taken as the standard of comparison. Moreover, if phthisis has any influence in causing epilepsy, we should expect to find it traceable more frequently in the cases without than in those with neurotic inheritance; but it is not so. Of 214 cases without, and 86 cases with, neurotic inheritance, the proportion of phthisis is just the same—39 per cent. Further, if phthisis exerted any causal influence, we should expect to find the proportion in which a neurotic heredity is traceable smaller when there was a tendency to phthisis than when there was not. This also is not the case. The proportion of inherited cases was rather less in these 300 cases than in the whole 1,250, being 28 per cent. The proportion was about the same in the cases with and those without a family history of phthisis, 28 per cent. in the former and 29 per cent. in the latter. These facts suggest that the association of phthisis and epilepsy is accidental. The same conclusion is suggested by a search for a history of epilepsy in the families of the phthisical. My colleague Dr. F. Roberts has been so kind as to question on this point several hundred patients suffering from phthisis, and the proportion in which any relative was known to have suffered from fits was only one in forty.

*Inherited Syphilis.*—In eight cases the patients were the subjects of well-marked inherited syphilis. In all of these cases the attacks had the aspect of idiopathic epilepsy, cases in which there were symptoms suggestive of local brain disease being excluded. In only two of these cases did the attacks begin in infancy. In all the others they commenced towards the end of or after childhood.

*Consanguinity of Parents.*—Enquiries were made in a considerable number of cases for consanguinity of parents, but in no instance was it met with.

*Age.*—We may consider next the influence of age on the occurrence of epilepsy, as evidenced by the date at which the first fit occurred in the series of 1,450 cases. If we take first decennial periods, we find that the number of cases in each period is as follows:—

| | Cases |
|---|---|
| Under 10 | 422 |
| 10–19 | 665 |
| 20–29 | 224 |
| 30–39 | 87 |
| 40–49 | 31 |
| 50–59 | 16 |
| 60–69 | 4 |
| 70–79 | 1 |

The percentage of the whole constituted by the cases beginning in each decennial period is shown in the following chart. Under ten years of age more than one quarter of the cases commenced, 29 per cent. Between ten and twenty nearly one-half of the total number, 46 per cent., began. In the next decennial period, twenty to thirty, the number falls to a seventh, 15·7 per cent. Between thirty and forty only 6 per cent. began, between forty and

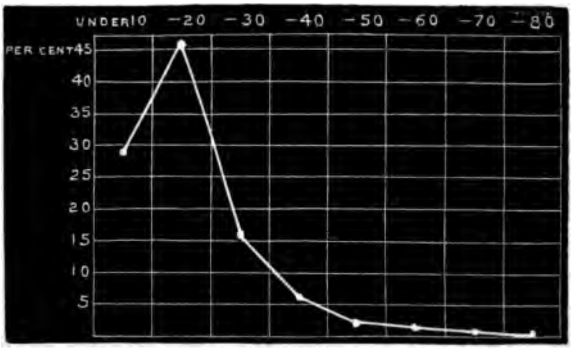

FIG. 1.—Proportion of 1,450 cases, commencing in each decennial period.

fifty only 2 per cent., between fifty and sixty only 1 per cent., and over sixty only ⅓ per cent. of the total number. Thus 75 per cent. of the cases commenced under twenty years. This proportion (1,087 of the 1,450) is exactly the same as that found by Hasse[1] in the largest collection of cases previously made (757 out of 995).

The relation of the cases to age is shown in greater detail in the next table, which presents the annual number of cases commencing in each year:—

| Age | Cases |
|---|---|
| Under 1 | 77 |
| 1 | 65 |
| 2 | 36 |
| 3 | 36 |
| 4 | 27 |
| 5 | 25 |
| 6 | 31 |
| 7 | 43 |
| 8 | 34 |
| 9 | 48 |
| 10 | 54 |
| 11 | 52 |
| 12 | 71 |
| 13 | 74 |
| 14 | 82 |
| 15 | 84 |
| 16 | 84 |
| 17 | 67 |
| 18 | 53 |
| 19 | 44 |
| 20 | 40 |
| 21 | 31 |
| 22–29 | 14–26 |
| 30–39 | 3–15 |
| 40–49 | 1–6 |
| 50–59 | 1–3 |
| 62 | 2 |
| 64 | 2 |
| 71 | 1 |

A large number of cases, no less than 12½ per cent. of the whole, commenced during the first three years of life. In this group no cases of simple infantile convulsions are included, only such as, beginning in infancy, continued as chronic epilepsy. There is a difficulty in ascertaining the exact date of commencement in all cases. Of about one-third all that could be learned was that they began in infancy. In the table these have been distributed through the first three years of life in the same proportion as presented by the two-thirds in which the exact date of commencement could be ascertained. The number of cases is largest in the first year (5½ per cent. of the whole), and falls rapidly to three years of age, and then more slowly until five, when the minimum for the early period of life occurs, only 1·7 per cent. commencing. From this there is a considerable rise at seven, the commencement of the second dentition, then a fall at eight, and from this the numbers increase slowly at nine and ten, rapidly at twelve, until the maximum is reached at fifteen and sixteen, at each of which 84 cases commenced, 5¾ per cent. of the total number. From this period we have a rapid fall to twenty-one, when only 2 per cent. of the whole commenced. During the next eight years the number of cases commencing in each year varied from 26 to 14. Between thirty and forty years the annual number varied between 15 and 3, and between forty and fifty it varied from 1 to 6. Between fifty and sixty the annual number of cases was from 1 to 3. Over sixty, the cases were—2 at sixty-two, 2 at sixty-four, and 1 at seventy-one.

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[1] Virchow's *Handbuch*, Abth. i., Bd. iv. p. 264.

*Relation of Sex to Age.*—The proportion in which the two sexes suffer varies considerably in the different periods of life. We may compare, first, their affection in the several decennial periods. In the cases commencing under ten years the females exceed the males by 6 per cent. In the second decennium, between ten and twenty, the excess of females amounts to 18 per cent. In the third, between twenty and thirty, there is a great fall, but the excess of females amounts to 12 per cent. Between thirty and forty, the numbers being still smaller, the relation is reversed; the males exceed the females by 16 per cent. Between forty and fifty, with a continued fall in numbers, the excess of males is still greater, amounting to 36 per cent. Between fifty and sixty it reaches 40 per cent., and over sixty males only suffer.

If we examine the several periods of life more minutely, we find that in the cases which commence in the first year of life the females are almost twice as numerous as the males (51 and 27). In the second year the number of females falls considerably, while that of males rises slightly, so that the difference between them is less. During the two following years the numbers fall, until a minimum of each is reached at five years, the females being still in slight excess. At six and seven years a rise occurs, and the number of cases in each sex is, in the latter, nearly the same. From seven to thirteen the earlier relation is reversed; the males are in excess of, or equal to, the females, with the perhaps accidental exception of the cases commencing at eleven. At twelve—that is, in the thirteenth year of life—the maximum for males is reached, and from this until sixteen there is a slight fall. The maximum for females occurs later than for males. The numbers increase at twelve and thirteen, and then more slowly, until at sixteen the maximum for any year is attained (50 cases), and they are in that year one-third more numerous than the males. During the next five years the numbers in each sex fall rapidly, the females being still in excess, but the two are nearly equal at twenty-one. The females continue, however, in slight excess until twenty-nine, when the two sexes are affected equally. After this the relation between them is reversed—in almost every year the males are more numerous. The females present no increase at the climacteric period, and they cease much sooner than do the males. The latest age at which epilepsy commenced in a woman was fifty-five, whereas in men cases commenced at fifty-eight, fifty-nine, sixty-two, sixty-four, and seventy-one. Thus at each of the maximum periods, at infancy and puberty, the excess of females is very great. During the later period of childhood the numbers are nearly equal. The excess of females lessens after puberty, and ceases by middle life.

*Relation of Heredity to Age.*—How far does an inherited tendency influence the age at which the disease commences? It is often said that when there is heredity, the disease begins before twenty. We shall, however, see that, so far from this being true, the influence of heredity continues until a late period of life, and, although absolutely greatest in youth, it is relatively only a little less during adult than during early life.

Particulars as to heredity and age at commencement were noted in 1,113 cases, of which heredity existed in

408. The distribution of these through life was nearly the same as that of the non-hereditary cases. About 5 per cent. began in the first year of life, and the annual number fell to a minimum in late childhood, to rise to a maximum at puberty. This maximum, however, occurred at fourteen years of age—a year earlier than the maximum for non-hereditary cases, which was at fifteen and sixteen. The cases with heredity lessen gradually during life, but continue to occur to the last. The latest case of the entire series was a man who suffered from what was apparently idiopathic epilepsy commencing at the age of seventy-one years, and in him the disease was inherited, his father having suffered from fits, clearly epileptic, for many years.

The relative frequency of heredity is shown by the proportion which the hereditary cases bear to the total number commencing at each period of life. The relative frequency, in periods of twenty years, is shown in the following table:—

| Age | Total cases | Heredity |
|---|---|---|
| Under 20 . . . | 844 | 319, or 37·8 per cent. |
| 20 to 39 . . . | 235 | 80, or 34 „ |
| 40 and over . . . | 34 | 9, or 26·5 „ |

The average for the whole of life is 36·6 per cent. This is exceeded by 1 per cent. in the first twenty years; in the second twenty years the percentage is only 2·6 below the average; while in the cases commencing over forty it is only 10 per cent. below the average.

*Relation of Heredity to Sex and Age.*—Since the first twenty years include the two maxima at infancy and puberty, it is desirable to examine the influence of heredity during this period in greater detail, and we may, at the same time, consider how far the two sexes exhibit its effects.

During the first three years of life, the disease is inherited by a larger proportion of females than of males (females 41, males 33 per cent.) During the next three years the proportion of heredity in females is nearly the same, that in males rises. The third triennial period, six to eight, presents a lower total percentage of hereditary cases than any similar period under twenty, but the minimum proportion in males occurs in the next triennial period, nine to eleven. In females, however, there is in this period a rise, and the maximum proportion for this sex is attained in the period twelve to fourteen; it falls considerably in the next two periods. The proportion in males rises after the minimum in the period nine to eleven, but never attains the proportion presented by the second triennial period of life. In the first twenty years as a whole, the excess of heredity in females over males amounts to 7 per cent. (males 34, females 41 per cent.) Between twenty and forty, the percentage heredity is nearly the same in males and in females. Over forty, of twenty-four males there was heredity in six; of seven females there was heredity in three.

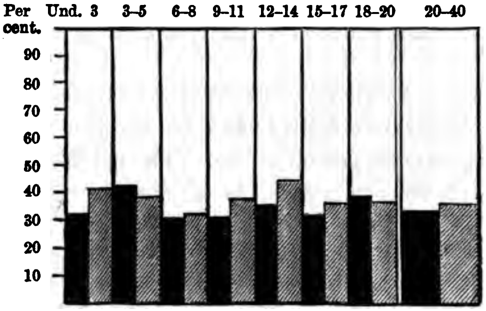

FIG. 2.—Percentage of hereditary cases in the first seven triennial periods, and in the period 20-40. The male cases are represented by the black, the female by the shaded columns. The fourth black column should be a little lower than the third.

*Relation of Form of Attack to Age and Sex.*—The next point which demands consideration is how far age and sex influence the occurrence of the hysteroid or coordinated forms of convulsion. We have seen that, of 1,000 cases in which the form of convulsion could be ascertained, it was coordinated or hysteroid in 185, or 18½ per cent. At every age the proportion of females is greater than of males. Under ten, these cases constitute 15 per cent. of the male and 18 per cent. of the female cases. Between ten and twenty the difference is, as might be expected, much greater. They constitute 14 per cent. of the male, and no less than 26 per cent. of the female cases. Between twenty and thirty the percentage of the males remains the same (14), and that of the females falls slightly (to 21). Between thirty and forty, the male cases with hysteroid symptoms fall to 12 per cent., while the females present a slight rise and constitute 24 per cent. In the male cases commencing over forty none of this character occurred; and in women, although 29 cases of the series commenced over forty, only 1 presented these symptoms. Thus cases with hysteroid symptoms commence almost exclusively under forty. In males, they bear at each age nearly the same proportion to the cases of pure epilepsy, decreasing very slightly from a maximum in childhood to a minimum in middle life. In women their minimum is in the first decade; their maximum is in the second; and the proportion after adolescence remains nearly double that of males during the period in which they occur. I think, however, that some surprise will be felt at the fact shown by these figures—which my actual observation of attacks fully confirms—that, even up to the fourth decade of life, one-third of the chronic convulsive cases presenting hysteroid phenomena (primary, or post-epileptic) occur in males.

### EXCITING CAUSES.

We must regard, as the exciting cause of epilepsy, the condition or circumstance to which the first fit was apparently due. It may be again pointed out, to prevent misconception, that these exciting causes cannot be regarded as the essential causes of the disease, except in a very small number of cases. They would, in most instances, have been ineffective had not a predisposition existed, due to inheritance, or to causes which are at present beyond our recognition. To use a familiar but useful illustration, the exciting cause, as a rule, bears no more causal relation to the disease than does a spark to the explosion of the gunpowder on which it falls. The real cause of the explosion is the chemical tension of the constituents of the gunpowder, the existence of which is revealed by the action of the spark. The real cause of the disease is the morbid state of the nervous system, the existence of which is only revealed by the effect of the immediate excitant of the first fit. Nevertheless, since the occurrence of one fit undoubtedly facilitates the occurrence of others, we must regard the excitant of the first fit as, to some extent, the cause of the other fits, *i.e.* of the whole developed disease. Without that cause, the disease might have remained for ever latent.

The study of these causes presents peculiar difficulty. The statements made by patients have, of necessity, to be received with caution, and the facts to be carefully investigated. An occurrence so striking as a fit is naturally assumed to be due to some cause at least discoverable, and so is often attributed to a remote and improbable antecedent. Of 1,150 cases in which this point was noted, a reasonable cause for the first fit was given in 428, or 37 per cent., and in the remainder no probable cause could be ascertained. From this series the cases distinctly originating in dentition convulsions are excluded.

*Relation to Sex.*—An exciting cause is met with in males much more frequently than in females: in 44 per cent. of men (235 out of 537 cases), and in only 31 per cent. of women (193 out of 616 cases)—a difference between the sexes of 13 per cent. This can scarcely be accounted for by greater exposure to exciting causes, since some of these act more on the female sex; it seems to indicate a greater predisposition on the part of women.

*Relation to Age.*—If we examine the proportion of cases in which an exciting cause can be traced in the different periods of life, we find that under ten it is nearly the same for both sexes (males 41, females 40 per cent.) From ten to twenty there is a great difference between the sexes, excitants being traceable in 19 per cent. more males than females (males 47, females 32 per cent.) In the next two decades, 20–40, the percentage in men continues nearly the same (at 42 per cent.); but in women it rises (to 35) in the first, and falls (to 32) in the second. In the cases which occur over forty years of age, the percentage in each sex is about equal (40); but in the male cases commencing over fifty, an exciting cause could be traced in as high a proportion as two-thirds. Thus the most striking peculiarity is the large proportion of cases which commence in women between ten and forty years of age without any discoverable exciting cause to which they can be ascribed.

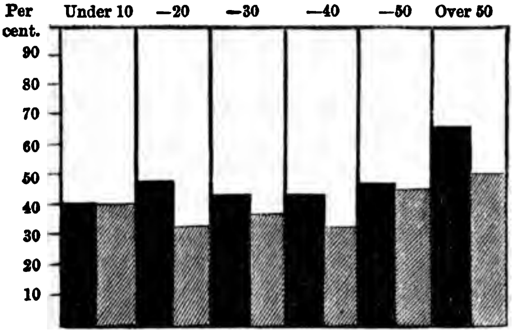

FIG. 3.—Percentage of 1,150 cases in which an exciting cause could be traced in each decennial period of life.

*Infantile Causes.*—Before considering in detail the several exciting causes which can be traced, it may be well to enquire what can be ascertained regarding the causation of the large number of cases in which the attacks commence in infancy, during the first three years of age.

Of these cases, 180 in number, no information was forthcoming in about one-third. In 24 cases, although information was obtained, no cause could be ascertained. In 98 cases, however, a cause for the first fit was assigned. In two, it was exposure to the sun; in one, ascarides; in seven, an acute disease; in eight, a fall; and in eight, the first fit occurred soon after birth, the labour in several of these cases having been difficult, and in some the forceps employed. In the remaining 72 cases, 33 males and 39 females, the first fits occurred during dentition, and were attributed to teething. The percentage of neurotic heredity in these cases was 34 per cent.—nearly the same as for the whole of life.

Thus nearly two-thirds of the cases beginning in infancy, the conditions of origin of which could be ascertained, arose from the so-called dentition convulsions. It seems legitimate to ascribe to the same cause a similar proportion of the cases respecting which no information was forthcoming. If so, we have a total number of cases due to this cause which constitutes 7 per cent. of all the cases investigated.

Few persons who have attended closely to the diseases of children can fail to be convinced of the truth of the opinion, long ago urged by Sir William Jenner, and now widely accepted, that almost all convulsions associated with dentition are really due to the constitutional condition of retarded development which we call rickets, *i.e.* to the irritability of the nervous system which accompanies this condition. The further details of the cases I am considering fully confirm this, for in a large number there was a history of late teething, late walking, and in many of crooked limbs.

Even these facts probably do not represent fully the influence of rickets in the production of epilepsy. In many of the cases just described, the course of the attacks was continuous from infancy to adult life. In others, however, there were intermissions, especially during the period of childhood. In several, for instance, the fits ceased at seven, to recommence at ten or eleven years. Now, in 27 other cases which commenced after the period of childhood was over, repeated convulsions had occurred during the first dentition, accompanied, in many, with other signs of rickets. It seems reasonable to ascribe to these convulsions of infancy a share in predisposing to the convulsions of later life, and the addition of these cases raises the number, in which a causal influence may with probability be ascribed to rickets, to nearly 10 per cent. of the whole.

Whether rickets is or is not entirely preventible, there can be little doubt that its development to the degree in which it leads to convulsions may always be prevented by proper attention to the diet and hygiene of infancy. These facts, therefore, suggest that a considerable proportion of the cases of epilepsy are really within the range of preventible diseases. They show also that inherited taint exerts the same influence here as at other periods of life, predisposing to the occurrence of convulsions in backward children, and to the persistence of the fits, and that the management of children with such inherited taint should be a subject of extreme care. Moreover, the occurrence of even a single fit in infancy indicates the need for future careful supervision.

Children, who have only an occasional fit during infancy and childhood on some considerable exciting cause, often become epileptic on reaching the epileptogenetic period of puberty. For instance, a child, without hereditary predisposition, had a fit at six months old, supposed to be due to teething (which was subsequently late), another fit at two years old during scarlet fever, another at 4½ during measles, and at 16½, after a carbuncle on the neck, he became permanently epileptic.

*Mental Emotion.*—Of all the immediate causes of epilepsy the most potent are psychical—fright, excitement, anxiety. To these were ascribed 157 cases, more than one-third of those in which a cause was given, and one-seventh of the whole series. Of the three forms of emotion, fright takes the first place with 119 cases, 10 per cent. of the whole. The relation of this cause to age is, however, very distinct. It is effective chiefly in early life, when emotion is so readily excited, and is most powerful at the transition from childhood to adult life, while after middle life it is almost inactive. Of the 119 cases only 3 commenced after thirty years of age, and 100 commenced under twenty. Of these, the majority, 70, commenced between ten and twenty, only 30 cases under ten. The female sex is notoriously the more emotional, and accordingly the disease, as Sieveking and others have pointed out, results from fright in a larger proportion of females than of males, although the difference is, perhaps, less than might be expected—71 females and 48 males; 60 and 40 per cent. respectively. It is notorious also that the difference between the sexes in emotionality increases as life advances. In childhood one sex is almost as emotional as the other, but with puberty men become far less emotional than women. The influence of fright as a cause of epilepsy is in strict harmony with this fact. Under ten years of age, the sexes suffer equally. Between ten and twenty the males are to the females as 3 to 4; between twenty and thirty as 3 to 13, and over thirty the only cases due to this cause occurred in women. After seventeen, only 3 cases occurred in men, while 20 occurred in women.

The influence of this cause is accurately recognised in popular phraseology, and is easily comprehensible when we consider how powerfully fright affects the nerve-centres. The stimulation of the motor centres shows itself in the sudden muscular actions, such as the start of alarm, which, no doubt, originates in the need for immediate action, which danger entails. The tremor, which often persists, indicates the enduring disturbance of the nerve-centres. In the organic nerves the vaso-motor centre is first stimulated and then inhibited, the sphincter centres are released from control, the sweat nerves are excited, and the heart's action is accelerated, perhaps by inhibition of the vagus. As Hughlings Jackson has put it: 'When we say anyone is frightened, we are asserting that there are occurring physical manifestations from discharges of the highest centres.'[1]

The exact form of fright varied, of course, in different cases, but the list teaches many lessons. In no less than eight cases the cause was some stupid practical joke—as a pretended ghost. In two cases children were shut up in dark cupboards. In five cases the fright was from an alarm of fire, in six from burglars. In six cases the patient had watched other persons in fits. Three resulted from alarm during severe thunderstorms. One case was that of a soldier who had his first fit a few hours after being terrified, while on sentry duty at night, by the unexpected appearance of some white goats on the top of the adjacent wall of a cemetery.

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[1] *Brit. Med. Journal*, January 25, 1879, p. 110.

Of 76 cases in which the interval between the fright and the fit was noted, the fit occurred immediately in 28, or one-third; within a few hours in 16. The interval was between a day and a week in 19, and it was more than a week in 13.

As might be anticipated, in a considerable number of cases the fits were of the hysteroid or coordinated type. Of 90 cases, in 61, or two-thirds, they were purely epileptic; in 29, or about one third, they were hysteroid. The difference between the two sexes in the frequency with which hysteroid fits succeed fright is not great. Of 51 cases in females, 18, or 35 per cent., were coordinated. Of 39 cases in males, 11, or 28 per cent., were of this type.

A relation can be traced between the interval that elapses before the fit and the character of the attack. The longer the interval the smaller is the proportion of coordinated fits. In the cases in which the interval was more than a week, only 18 per cent. were of this character; in the cases in which the interval was less than a week, but more than a day, the percentage of this form was 21. In the cases in which the interval was only a few hours the percentage was 25, while in those in which the fit occurred immediately it was not less than 57.

Other forms of sudden emotional excitement caused the first fit in 19 cases—9 males and 10 females; and these cases also occurred during the emotional period of life, under thirty years of age.

*Prolonged mental anxiety* is, in some cases, the only influence to which the disease can be assigned. In this series of cases it was given as the cause with apparent reason in 29 cases, in which males preponderated. This cause is effective late in life more frequently than most other causes, and is especially active in men; seven of the cases were over thirty years of age, and of these six were males.

*Traumatic Causes.*—Next, in numerical importance, among the exciting causes of epilepsy are traumatic influences, blows and falls on the head. To these, after the elimination of doubtful instances, 65 cases were due. A third (21 cases) occurred under ten years, rather more than a third (25 cases) between ten and twenty. Males are affected by this cause more than females, the numbers being 39 to 26. This is clearly due to the risks of occupation, for in the cases commencing before ten years the females were in excess. Between ten and forty the male cases in each decennium were rather more than twice the number of the females. Over forty the only cases were in men.

Hysteroid attacks rarely result from a traumatic cause. They were met with in only 6 cases out of 44 in which the character of the fit could be determined; and in estimating the significance of these, it must be remembered that traumatic causes often involve intense emotional disturbance.

In two-thirds of the cases the injury was a fall on the head; in one-third it was a blow on the head. In most of the cases the patient was stunned for a time, but in only a fifth (8 cases) did the first fit occur immediately. In the rest an interval elapsed; in one-third of the cases the interval between the injury and the fit was more than a day and less than a week; in another fifth the interval was between a week and a month, and in about the same proportion the interval was more than a month.

It may be urged that these traumatic cases should not be included among cases not due to organic disease. But it is certain that a blow or fall may excite fits without causing any visible lesion of the brain, and all cases were excluded in which the present or past symptoms, or the mode of onset of the fits, made it probable that 'coarse' changes had been produced.

*Exposure to the sun* is frequently assigned as the cause of the first fit, but the relation is often doubtful. Sunstroke affords so ready an explanation of a convulsive seizure occurring in the hot sun, that it is naturally regarded as the cause of the attack in all such cases. It is an undoubted cause in a few. After the elimination of all doubtful instances, in which some other cause could be traced or in which the heat of the sun was not very intense, or the exposure prolonged, there remained twenty seven, in which it seemed probable that the first fit was due to this cause. In 10 of the cases there was a distinct attack of sunstroke, occurring, in 6, in the tropics. From this cause, as may be expected, males, from their greater exposure, suffer most: 20 were males, and only 7 females. The cases were pretty equally distributed through life, but those in females were relatively most numerous in childhood, and decreased in adult life, to cease at thirty. The form of convulsion was almost always pure epilepsy, only one case presented hysteroid symptoms. In 10 the fit occurred immediately; in several others, in which an interval elapsed, this was marked by some form of nerve disturbance, such as tremor.

*Acute Diseases.*—Recurring epileptic convulsions sometimes succeed acute specific diseases. In some of these cases the convulsions are distinctly 'post-hemiplegic;' an attack of paralysis occurred, probably from thrombosis, during the illness, and this was succeeded by recurring convulsions. In other cases there is no history or indication of any preceding paralysis; the convulsions are apparently unconnected with any organic brain lesion. This was the case in thirty-seven cases (14 males and 23 females). The diseases, in the course of or after which the first fit occurred, were ascertained in 35 cases, and were as follows:—

| | Cases |
|---|---|
| Scarlet fever | 19 |
| Measles | 9 |
| Fever, 'low,' 'typhus' | 5 |
| Rheumatic fever | 1 |
| Diarrhœa | 1 |
| | 35 |

The remarkable influence of scarlet fever in the causation of epilepsy demands further consideration. The cases due to this cause commenced during early life, under seventeen. It may be thought that the first fits in these cases were due to uræmic poisoning, but this is not supported by the particulars ascertained regarding 14 cases. It is true that in three of these all that could be learned was that the first fit occurred during scarlet fever accompanied with dropsy, and in two others that the fits occurred after an attack of scarlatinal dropsy was over. But in two other cases in which the scarlet fever was succeeded by dropsy, the first fit occurred after the fever, before the dropsy. In six cases there was no dropsy; in two of these the first fit occurred after the fever was over, in one during convalescence, and in one a month later. In three cases the first fit occurred during the height of the fever, and in one convulsions ushered in the disease, persisted throughout the attack, and continued afterwards as permanent epilepsy. In only one case did ear disease also result from the fever. The urine was examined in most cases, but no albumen was found.

These facts suggest that the first fits were due to some peculiar effect of the scarlet-fever poison upon the nervous system. It may be remembered that the specific poison has a very widespread action in the system, and that, after scarlet fever, optic neuritis, leading to subsequent atrophy, may occur, without renal or coarse cerebral disease.

*Intestinal Worms.*—Acute convulsions frequently result from the irritation of various forms of intestinal worms in children, and sometimes in adults. Usually, however, they cease when the worms are expelled. In rare cases, the attacks, set up in the first instance by the intestinal irritation, recur and continue after the irritation is at an end. In six cases the first fit was apparently due to this cause; the attacks had continued, although the worms had been expelled, and a renewal of the vermifuge treatment had no influence on the disease.

*Digestive derangement* is an occasional cause, and was noted in five cases in the series. Although it is not often that the first fit can be ascribed to gastric derangement, yet dyspepsia is common in epileptics, and, as Dr. Paget of Cambridge has especially insisted, often excites attacks in those subject to them.

*Asphyxia.*—In nine cases immersion in the water caused the first fit. This is an influence which involves both fright and asphyxia, but the influence of the latter was well shown in another case. A child, æt. 3½, tried to swallow a large piece of potato, which stuck in his throat and stopped his breath. He became livid, unconscious, and convulsed before the obstruction was dislodged with a spoon. The convulsive twitching continued for some minutes, and twenty minutes passed before he regained consciousness. Three days afterwards he had another fit, and they continued until he came under treatment, several months later.

*Chronic alcoholism* is a rare cause of epilepsy.[1] Convulsions not unfrequently result from it, but usually in association with distinct symptoms of chronic meningitis. Now and then, however, simple epileptiform convulsions apparently result and exist without other sign of brain-mischief. It was the apparent cause of 13 cases only in the series. In some the first fit occurred during intoxication, and in a few the attacks recurred after each alcoholic excess. Epilepsy from absinthe-drinking, occasionally met with in France, is unknown in this country.

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[1] The conclusions from my own experience do not accord with those of Echeverria (' On Alcoholic Epilepsy,' *Journal of Mental Science*, Jan. 1881).

*Lead Poisoning.*—Six patients were the subjects of chronic lead-poisoning. In two of these there was also chronic renal disease. In one of the others the epilepsy had existed from infancy, but became much more severe when the plumbism was developed. In the remaining three, the lead-poisoning was the apparent cause of the attacks, which resembled, in character and course, those of idiopathic epilepsy. It is well known that acute lead-poisoning may be accompanied by convulsions; that chronic poisoning may cause chronic convulsions precisely like those of ordinary epilepsy, has been pointed out by others, but is not, I think, generally known. The list of nervous disturbances which may result from lead is a long one, and includes, as cases I have seen testify, acute and chronic mental derangement, neuralgia, brachial spasm as well as palsy, general muscular wasting and optic neuritis. It is known that lead is to be found, after death, in the brain in considerable quantity.

*Renal Disease.*—I have mentioned that in two of the cases of lead-poisoning there was also consecutive renal disease. In two other cases of chronic renal disease convulsive attacks, resembling perfectly idiopathic epilepsy, brought the patients under treatment. In one the absence of any coarse changes in the brain was proved *post mortem.* It appears, therefore, that such convulsive attacks may occur and recur for months, without other sign of uræmic mischief. Several cases in which chronic mental derangement resulted from Bright's disease have also come under my notice.

*Tobacco.*—I have met with no case which suggests that tobacco-smoking exerts any influence in the production of epilepsy; but in one case the fits were apparently due to the patient, a lad, having shortly before commenced working in a tobacco factory, where his occupation caused frequent nausea.

*Anæsthetics.*—In one case the first fit occurred after the administration of chloroform, and, in one, attacks, which had ceased for many years, recurred after the inhalation of nitrous oxide. It is remarkable that when the anæsthesia from nitrous oxide is passing off, transient hysteroid convulsion frequently occurs. The legs and arms are stretched and stiff, sometimes quiver, the patient looks wild, talks for a moment as if dreaming, then passes into sleep for a few minutes, and is better.

*Disturbed Menstruation.*—Retarded or absent menstruation coincided with the first fits in a large number of the cases which commenced in girls between fourteen and seventeen, but the difficulty in determining the exact causal relationship between the two conditions is very great. Epilepsy once set up in such cases, the subsequent establishment of regular menstruation appears to exercise very little influence upon the fits.

*Pregnancy.*—The first fit occurred, in seven cases, during pregnancy, without other obvious cause, and in five cases the disease commenced after parturition.

*Masturbation.*—Many circumstances render it very difficult to determine the influence of masturbation as a cause of epilepsy. The habit is common in epileptic boys, as in others, but we cannot infer that, in all such cases, it is the cause of the disease. The etiological relation can only be regarded as established when the arrest of the habit, as by circumcision, arrests the disease. But the converse is not true; the continuance of the disease after the arrest of the practice does not disprove the relationship, because, when the 'convulsive habit' is established, it frequently persists after its cause has ceased to be effective. Moreover, in private, it is often difficult to ascertain the existence of the practice, for it is remarkable how long it may elude the observation of friends. It is usually denied by the patient, and the very enquiry renders its discovery more difficult by suggesting the necessity for concealment. I am inclined to think that it is much less frequently a cause of true epilepsy than of untypical attacks, sometimes hysteroid, sometimes of characters intermediate between hysteroid and epileptoid form. I have so frequently in boys met with this form of attack in association with the practice, that I can scarcely doubt their etiological connection.

*Syphilis.*—Convulsions are very common in cases of syphilitic brain disease, syphilitic tumour, and chronic meningitis. Such convulsions may recur in chronic course, and may persist after the original disease has been rendered quiescent by treatment. The cases which present them are often called 'syphilitic epilepsy.' But they differ pathologically from the cases to which the term 'epilepsy' is strictly applicable, in that *post mortem* there is to be found visible organic brain disease—chronic meningitis or growth. In most cases the convulsive attacks have the deliberate march and limited range characteristic of those due to organic brain disease, of which there are usually other indications.

Does syphilis cause epilepsy independently of the agency of organic brain disease—by such an action of the syphilitic poison upon the nervous system as eludes discovery by the most careful naked eye and microscopical investigation? Fournier[1] has maintained that it does; that in the early period of constitutional syphilis a morbid state of the nervous system is induced by the influence of the syphilitic poison; and that this morbid state may be manifested by various functional derangements, as epilepsy, hysteria, and chorea. He asserts that 'secondary' epilepsy is without visible lesions, while 'tertiary' epilepsy results from organic disease. Stated thus, the assertion is unquestionably inaccurate. My own experience entirely agrees with the statements of Echeverria,[2] that organic disease, especially meningitis, is not rare in the secondary period, and that, in most cases of convulsions occurring during the secondary period, symptoms of such organic disease may be found. A few cases commencing in the secondary period have the aspect of idiopathic epilepsy, but in most of these, other causes, such as inherited predisposition, can be traced, and it seems unjustifiable, in an enquiry into causes, to regard the coexistence of syphilis as more than a coincidence in such cases.[1]

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[1] *Ann. de Dermatologie et Syph.* 1880, pp. 160, 199.

[2] *Journal of Mental Science*, July 1880.

There remain, however, a very few cases in which no other cause can be traced, and in which, when iodide of potassium alone is given, the convulsions cease. Most of the cases which have been recorded in proof of the existence of such true syphilitic epilepsy are inconclusive: either the symptoms of the case, and the mode of onset of the fits, are so imperfectly described that the existence of organic disease cannot be excluded; or there existed other sufficient cause for the attacks; or bromide of potassium was combined with the iodide, thus depriving the effect of treatment of its significance. Cases which are not open to these objections are extremely rare, and I have only met with one. It was that of a man, aged 37, who had a primary sore a year previously, subsequently sore throat, and presented traces of past iritis. There was no neurotic family history. Nine days before being seen he had a severe fit, without warning, in which he bit his tongue. There was no headache, ophthalmoscopic change, or other indication of organic brain disease. Ten grains of iodide of potassium, three times a day, were ordered, and four months later, when last seen, he had had no recurrence. Even such a case, however, is rendered scarcely conclusive by the fact that occasionally organic brain disease may cause no symptom but convulsion; even, as I have seen, headache may be absent; and such convulsion may have the sudden general character met with in idiopathic epilepsy. The question must thus be regarded as still an open one. The evidence that idiopathic epilepsy may result from syphilis is certainly far slighter in the case of the acquired than in that of the inherited disease.

The associations of epilepsy with heart disease, with chorea, and with past, inactive organic disease of the brain, are considered in another chapter.

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[1] It is not denied that if constitutional syphilis acts as a cause of epilepsy independent of organic disease, it may be effective in patients with inherited tendency, the disease being the result of both influences. But in an enquiry to ascertain the facts, such cases should be excluded.

In this account of the causes of epilepsy, no mention has been made of one to which a prominent position is usually assigned—the irritation of a peripheral nerve, as by a foreign body, a spiculum of bone, or a decayed tooth. The occurrence of convulsions from this cause is proved by the testimony of good observers, but such cases are extremely rare, and no instance has come under my own notice.

# CHAPTER II.

## *SYMPTOMS.*

### *GENERAL CHARACTERS OF EPILEPTIC FITS.*

THE attacks or seizures which characterise epilepsy are commonly divided into two classes, major or severe, and minor or slight. These two forms, although clearly distinguished in their general characters, are not separated by a sharp demarcation. In the major attacks (*grand mal*) there is loss of consciousness, often prolonged, and severe muscular spasm. In the minor attacks .(*petit mal*) there is commonly brief loss of consciousness without muscular spasm; sometimes there is loss of consciousness and slight muscular spasm; very rarely there is slight muscular spasm without loss of consciousness.

In severe attacks the patient, if standing, falls to the ground; in slight attacks he may or may not fall. Hence the term 'epilepsy,' equivalent in meaning to the old English name, 'falling sickness,' is founded on a feature which is not invariable.

In very severe attacks muscular spasm comes on at the same time as consciousness is lost. In less severe fits the muscular spasm may commence before consciousness is lost, and the patient is then aware of the onset of the fit. Still more frequently the spasm and loss of consciousness are preceded by some sensation. The sensation, or commencing spasm, which informs the patient of the oncoming attack, constitutes the 'warning' or 'aura' of the fit.

*Major Fits.*—At the onset of the severe fit the spasm is tonic in character, rigid violent muscular contraction, fixing the limbs in irregular positions. There is usually deviation of the eyes and rotation of the head towards one side, and this rotation may involve the whole body, and sometimes cause the patient to turn round, even two or three times. The tonic spasm involves the muscles of the chest and abdomen. The features are distorted; the face, usually at first pale, becomes suffused and then livid, as the chest is fixed and respiratory movements are arrested. The eyes are open or closed; the conjunctiva is insensitive; the pupils dilate widely as cyanosis comes on. As the spasm continues, it commonly changes in its relative intensity in different parts, so that slight changes in the position of the strained limbs occur. Presently, when the cyanosis has become intense, the fixed tetanic contractions of the muscles can be felt to be vibratory, and the vibrations increase to slight visible remissions. As these remissions become deeper the muscular contractions become more shock-like in character, and the stage of clonic spasm is reached, in which the limbs, head, face, jaw, trunk are jerked with violence. In the resulting movement of the chest, air is expelled from the thorax, and bloody saliva is frothed out between the lips. The air entering the lungs is at first insufficient to lessen the lividity, and the patient may seem to be at the point of death. But as the intervals between the shocks of spasm lengthen, and the remissions become greater, more breath enters the chest, and the lividity lessens. In becoming less frequent the muscular contractions do not become less strong, and the last jerk is often as violent as those which have preceded it. At last the spasm is at an end, and the patient lies senseless and prostrate, and usually sleeps heavily for a time, and then can be roused. Urine frequently, and fæces occasionally, are passed in the fit.

In some cases the spasm is more deliberate in its onset. Instead of commencing simultaneously in all the muscles of the body, it begins in one region, as the face or arm, and thence spreads, first to the limbs on the same side, the head and eyes being turned towards that side, and then, lessening on the side first affected, it invades the limbs on the other side, with a corresponding rotation of the head. Such attacks may commence with tonic spasm; less frequently they commence with, and consist of, clonic spasm only. This form of convulsion is that which is most common in organic cerebral disease, such as tumour, but it is also often met with in idiopathic epilepsy. In such cases, consciousness is often lost late, so that the patient is aware of the commencing spasm.

*Minor Attacks.*— The slight attacks of epilepsy may be characterised by loss of consciousness only. An individual suddenly stops in his occupation, looks strange for a moment, perhaps turns pale, and then goes on with what he is doing, may even finish a sentence which he had commenced, and be aware that something has happened only by finding that he has dropped what was in his hands, or that persons near are looking at him in surprise. The attack is, however, often heralded by some sensory warning or aura, such as precedes the major attacks. It may be accompanied by slight visible spasm, such as putting the arms forward and bending the head down, or there may be slight convulsion in the part in which the spasm commences in the severe attacks, and, in some cases, the minor attack may be constituted by such spasm, without loss of consciousness. After a slight attack is over, the patient may be quite well. Often he is stupid and dull for a time, and sometimes proceeds to perform some action in a dreamlike, automatic manner, such as undressing himself, retaining afterwards no recollection of what he has done. Sometimes this stage is marked by passion and violence.

After an attack, major, or more frequently minor, instead of presenting an automatic state, the patient may pass into a condition of hysteroid or coordinated convulsion, such as has been mentioned at p. 2, and will be more fully described in a future chapter. In some patients in which this sequence occurs, all the obtrusive phenomena of the attacks may be of this hysteroid character.

### *SYMPTOMS IN DETAIL.*

Some of the symptoms of attacks may now be considered in greater detail. In describing them, however, it will be well to point out some of the physiological and pathological facts which underlie them, where these can be traced. By doing so, the symptoms themselves are rendered more intelligible, and their study more instructive, than if they are merely enumerated without reference to their pathology and pathological physiology. The method here followed involves the assumption of some conclusions, which an attempt will be made to prove in the chapter on 'Pathology,' especially in relation to the part played by the cerebral hemispheres in causing the phenomena of attacks. It is also necessary, in discussing the symptoms of idiopathic epileptic fits, to allude to the characters of the analogous convulsive seizures which result from such organic diseases of the brain as tumour. It will, however, be understood that none of these cases are included in the statistics given.

### PRECURSORY SYMPTOMS.

Besides the immediate warning, attacks in some patients are preceded for some hours or days by symptoms indicative of slight disturbance of the nerve centres, and by these, the patients and their friends know that an attack is impending. One of the most frequent of these precursory symptoms is the occurrence of sudden starts or jerks, affecting both sides, sometimes the arms only, sometimes the legs also. The patient may, in the jerk, drop anything which is in the hand, or even, when the legs are affected, may fall down. These jerks occur in some patients for an hour or two, in others for as long as a week, before a fit. They are usually limited to the waking state, but in rare cases occur only during sleep, or during the state of 'going to sleep.' Another occasional symptom of this kind is giddiness, not paroxysmal, as in *petit mal,* but more or less constant. It usually precedes a fit for a few hours only, but in some cases for a longer time. One patient, whose fits were at long intervals, was always giddy for a week before an attack. A vague sense of dyspnœa existed for an hour before every attack in another patient, the immediate warning being a sudden pain in the head, and the fit epileptic. Occasionally the precursory symptom is an unnatural appetite. The patient eats ravenously, and never seems satisfied, and from this the friends know that an attack is impending. In another patient, in whom also the immediate warning was pain in the head, each fit was preceded for some hours by giddiness and loss of sight. Occasionally (as Aretæus remarked) flashes of light or colour are the precursory symptoms, as in a case in which the fits began in the leg and were preceded for some hours, or even days, by occasional 'sparks of red fire' before the eyes. Often there is an altered mental state, such as irritability of temper, by which the friends of patients become aware that, as they say, 'a fit is about.' It will be seen from the facts mentioned that these precursory symptoms are not usually of the same character as the immediate warnings of the fits.

In rare instances the onset of the fit is preceded by some automatic action, as running. One patient always ran a short distance, and then fell in the fit. These cases were termed 'epilepsia cursiva' by Bootius.[1] Another patient, whose attacks were epileptic and severe, if walking, always turned back and walked back a short distance, and then fell in the fit. He turned back in the same manner although he was going home, and near home.

The face usually becomes pale at the onset of a fit. This symptom is not, however, invariable, and undue stress has been laid upon it. In some cases the face is flushed at the onset, and afterwards becomes pale.

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[1] In *Observationes medicæ de Affectis omissis.* Lond. 1649. Several instances were described by Andree in *Cases of the Epilepsy*, &c. 1746.

### MODE OF ONSET—WARNING—AURA.

The information which is furnished by the modes in which attacks commence is of great importance, practical and pathological. It is only of late that its importance has been recognised. The convulsions in idiopathic epilepsy are so rapid in their evolution, and so similar, in most cases, in their developed characters, that mere observation of attacks was for a long time unproductive of much addition to our knowledge. A new era in the study of epilepsy may be said to have commenced with the investigation, by Hughlings Jackson,[1] of the modes of onset of attacks which begin deliberately, combined with that of the subjective sensations with which attacks commence that are less deliberate in their onset—a mode of investigation which, much more than the attacks themselves, deserves the epithet 'Jacksonian.'[2]

In considering this part of the subject it is important to distinguish, but not to separate, the cases in which the visible phenomena of convulsion are epileptoid, and those in which they are hysteroid. The conjoined study of the modes of onset of these two forms of convulsion is inevitable in a practical discussion of the subject.

The aura of an attack, *i.e.* the sensation, usually in some part of the periphery, with which attacks commence, was formerly regarded as an actual process taking place in the part to which the sensation was referred, and commencing there. The word 'aura' was first used by Pelops, the master of Galen, who was struck by the phenomena with which many attacks begin—a sensation, commencing in the hand or the foot, apparently ascending to the head. The sensation having been described to him by patients as a 'cold vapour,' he suggested that it might really be such, passing up the vessels, then believed to convey air. Hence he termed it *πνευματικὴ αὔρα*, 'spirituous vapour.'[1] This notion of the peripheral origin of the aura was maintained until recent times, but with the discovery of the functions of the vessels and nerves, its seat was transferred to the latter; and the theory was held to be confirmed by cases to which far more significance was attached than their extreme rarity warranted, in which such local aura was associated with permanent lesion and irritation of a nerve in the part. It is now known, however, that the local aura is, in the vast majority of cases, merely the result of the commencing process in the brain, either affecting the consciousness directly, and causing a sensation which is referred to the part, or producing local spasm, and thus affecting the consciousness indirectly. That the aura is merely the commencement of the fit, and not its cause, although not generally recognised until the present century, was pointed out three hundred years ago by Erastus.[2]

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[1] Especially in 'A Study of Convulsions,' *Transactions of the St. Andrew's Med. Graduates' Association*, vol. iii. 1870; and 'Localisation of Movements in the Brain,' *Lancet*, 1873, vol. i.; also republished separately.

[2] However anxious to do honour to a distinguished fellow-countryman, I cannot think it desirable to add to the number of diseases and symptoms, already too great, called after the names of observers—a system of nomenclature alike perplexing to students, confusing to workers, and unscientific in principle.

The fact that an aura commencing in a limb may be arrested by a ligature around the limb, above the place in which the aura is felt, was once thought to constitute evidence that the process of the convulsion originated at the periphery. It is now known that the ligature will arrest a fit which is due to a cerebral tumour as effectually and more frequently than one due to any other cause. This was first pointed out in the beginning of this century by Odier.[3] It has been insisted on by Brown-Séquard, and I have seen many striking instances of it.

When the aura is regarded as the expression of the commencing change in the brain, its study becomes of even greater importance than when it is regarded as originating at the periphery. By this means we gain information, to be obtained in no other way, of the brain function first deranged—that is, of the functional region in which the process of the fit begins. In some cases, at least, this may be regarded as the seat of the disease. We learn also, as Hughlings Jackson has so well shown, something of the form of normal cerebral action which disease modifies, rather than subverts.

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[1] Galen, *De locis affectis*, lib. iii. cap. 11.

[2] Erastus, *Disput. de Med. Nov. Paracelsi* (circa 1580).

[3] Odier, *Manuel de Méd. Pratique*, Geneva, 1811.

The abnormal action of the nerve centre, by which the phenomena are immediately produced, may be conveniently spoken of (following the example of Hughlings Jackson) as a 'discharge.' The word is thus employed merely as a designation for the sudden violent functional activity of nerve cells, causing the spasm which the observer sees, or the sensation of which the patient is conscious. Thus used it involves no theory of the nature of the process which goes on in the nerve cell. It merely designates that which no one can deny, the sudden liberation of energy somewhere in the nerve centres. The energy liberated is what we call 'nerve force,' which may excite another liberation of energy in the muscles, as visible spasm. The liberated nerve force is thus recognised by another person solely by its effects, but it may directly affect the consciousness of the individual. Hence the study of the subjective modes of onset of fits, the warnings of which the patient is conscious, is of great importance and full of promise of additions to our knowledge of the intimate pathology of the disease.

The aura of an attack, using the term in its widest meaning, as signifying the subjective commencement, may consist in a consciousness of motion, or of sensation, or in a mental state. It has been long and often maintained by Hughlings Jackson, in extension of the older view of Laycock, that the whole brain is made up of structures which subserve sensori-motor processes, and that into such processes all its functions may be resolved. It is unnecessary now to consider this view as a whole, but that every structure of the brain concerned with sensation proper is connected, directly or indirectly, with a part concerned with motion, may be regarded as a proposition scarcely needing proof. A discharge, such as occurs in an epileptic fit, taking place in either of these related structures, may remain confined to that in which it commenced, but it usually leads to a discharge in the other. The two may act so simultaneously that the resulting motion and sensation occur together. The patient may feel a tingling in his hand at the same moment as the fingers begin to twitch. Or the discharge in one may lead the way—be followed by the other at an interval. The twitching or the tingling may come first. I think that it is of importance to determine, in any given case, with which the attack commences—the motion or the sensation—*i.e.* whether the motor or the sensory centre leads in the discharge. By so doing we are able to understand better the association of auræ and their progress. It is necessary, however, carefully to discriminate the form of the sensation, because, as Hughlings Jackson has also pointed out, the consciousness may be affected directly by the action of a motor centre, although no movement results. There are motor sensations as well as sensory sensations, and, as we shall see, a fit may begin by a sense of motion before any actual movement occurs, and the sensation of motion may be vastly in excess of the real movement. The word 'sensation,' however, will here be used, when unqualified, to signify a sensation proper, such as numbness, tingling, and the like.

*Frequency of Warnings.*—In what proportion of cases is consciousness lost so early that the commencement of the fit is unfelt? Of 1,000 cases, in which the presence or absence of a warning was noted, it was always absent in 495, while some aura existed, at least occasionally, in 505. Thus, roughly speaking, loss of consciousness precedes or accompanies the first symptoms in half the cases; in the other half the patient is aware of the commencement of the attack. This proportion agrees closely with that ascertained by Romberg and Sieveking. We may consider what facts regarding the modes of onset these 505 cases supply.

FORMS OF AURA.—The sensation or motion with which the attacks commence may be referred to almost any part of the body, limbs, head, trunk, organs of special sense, and to many viscera. We must therefore infer that the process of the fit may commence in any part of the brain in which these various parts are represented. We may, for the present purpose, classify most of the auræ or modes of onset into seven groups: (1) The unilateral auræ, a motion or sensation in one side of the tongue, face, trunk, or in one arm or one leg. (2) Certain general auræ; bilateral sensations in the limbs, tremors, starts, malaise, faintness, &c. (3) Auræ referred to certain organs, mainly to those to which the pneumogastric nerve is distributed, and to this group belong most of the visceral warnings. The most common is the well-known epigastric sensation, and others are a feeling of choking, dyspnœa, nausea, and cardiac sensations. (4) Vertigo and other allied sensations. (5) Certain sensations in the head, pain, &c. (6) Psychical auræ, the consciousness of an emotion or an idea. (7) Special sense warnings; some of these are strictly unilateral, but it is convenient to consider them together. This classification is clinical, and as such is adopted as affording facilities for the determination of facts apart from theories. Our knowledge of the significance of many warnings is still too imperfect to permit an exact scientific classification to be framed.

*Unilateral Commencement.*—We may take first the unilateral peripheral auræ, present in 86 cases, or 17 per cent. of those with warnings.[1] Of these the commencement was in the arm in more than half (45 cases), in the leg and face in smaller and nearly equal numbers (15 and 17 cases), in the tongue in five, and in the side of the trunk in only two cases. Such unilateral commencement is common in seizures due to organic brain disease, and the organic disease causing them is usually on the surface of the brain, in the region stimulation of which causes movements in the parts first affected in the fit. Such stimulation, indeed, as Ferrier has shown, may cause convulsions beginning in the part. But from the series of cases now under consideration all those presenting symptoms suggestive of organic brain disease were excluded.

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[1] The unilateral commencement attracted much attention from the time of Aretæus, and Herpin (*loc. cit.* p. 389, *et seq.*) has collected a series of instances from the writings of Aretæus, Galen, Alexander Trallianus, among the Greeks, and Ali-Rodoham, Brassavola, Sylvius, Faventinus, Dovinctus, and Hollier among the writers of the sixteenth century, of cases commencing by an aura in the hand, foot, calf, face, &c. They were also studied by Bravais in 1824, but were not, as is sometimes erroneously stated, first described by him. They have been most carefully investigated by Hughlings Jackson, to whom our present knowledge is largely due.

In five cases only the aura was referred first to the tongue, in two others the tongue was affected secondarily. In one the commencement was by a movement, in one by a beating, in the rest there was merely a sensation; in three tingling, and in one a sensation of something crawling on the tongue. In each case the convulsion was right-sided, and in each case in which the side of the tongue affected was noted, this was also the right side. Hence it seems probable that the sensori-motor processes for the tongue predominate on the left side of the brain, a fact also suggested by the great impairment of the movements of the tongue frequently noticed in right hemiplegia with aphasia.

The association of movement of the tongue is twofold, one in its use for speech, one in its use in mastication and deglutition. We may trace this double association in the combinations of the aura. Tingling in the tongue was associated in one case with twitching in the lips, in another with a lateral movement, or sense of movement, in the jaw. The sensation of something crawling on the tongue, in one case, was followed by a feeling of sickness, then of something rising in the throat, and then by palpitation of the heart.

The seizure commenced in the face in 16 cases, and on one side as frequently as on the other, but much more frequently by a motion than by a sensation. In eight cases the mouth was drawn to one side, no doubt by the action of the zygomatic muscles. Commencement in the lips was rare. In two cases there was an initial contraction in the orbicularis palpebrarum, and in both cases this was associated with a sensation in the right hand, and the fits were right-sided.

In most cases of organic disease in which the convulsion commenced in the face, the lesion has been found to occupy the third frontal convolution. This convolution on the left side is also connected with voluntary speech. Hence we find that inability to speak often accompanies a fit which commences in, or early involves, the right side of the face. For instance, a girl had minor seizures, which consisted in a sensation of tingling in the right arm, which passed up to the angle of the mouth, and then she then became unable to speak for about five minutes. In left-handed persons the 'speech-centre' is usually on the right, and not on the left, side of the brain, and the association just mentioned was well exemplified by a left-handed man, who, at the age of thirty-one, became liable to fits which commenced by spasm in the left side of the face, spreading thence to the left arm, with loss of consciousness. Inability to speak preceded each attack for ten minutes, and persisted afterwards for the same time. Some of these cases are accompanied by a simultaneous sensation in the tongue.

The cases in which the attacks commenced in the arm were forty in number. In about three-quarters of the cases the commencement was in the hand. Of those beginning by spasm, twitching, &c., the first motion was, in most instances, either in the hand as a whole (ten cases) or in the arm as a whole (eight cases). In only one case was there initial movement in the fingers, and this was clonic spasm in the common flexors. Rarely the spasm begins in the shoulder, as in a case in which the attack was carefully watched, and the movement distinctly descended the arm. In 14 cases the attack began by a sensation— 'numbness,' 'tingling,' 'pins and needles,' &c.;

and in these the commencement was, in most, in a definite part of the hand—the forefinger in three cases, never in the thumb, twice in all the fingers, once in the middle finger, once in the palm, twice in the back of the hand, three times in the hand as a whole, and twice at the wrist. It never began higher up the arm. In five other cases the sensation with which the fits began was less simple, and in several was certainly a motor sensation. One patient always felt 'as if his arm were drawing up,' and would beg that it might be held down, although there was no motion in it. The feeling was accompanied by severe pain 'as if the arm would break.' In another case there was a somewhat similar pain, 'as if the arm were withering up.' In a third there was a feeling of stiffness, and in a fourth a feeling of twitching in the first three fingers, apparently without movement, and in a fifth there was a sensation in the back of the arm 'as if the nerves were being drawn.' In another patient the attacks began by twitching in the thumb and forefinger, visible to others; the arm then dropped by the side, but always seemed to the patient to be raised up over the head by spasm.

The attacks beginning with distinct spasm, or with the sensations last described, probably motor, began in the right and left hand in an equal number of cases, but those commencing with a simple sensation began in the left hand twice as frequently as in the right.

One variety of commencement in the arm deserves special mention on account of the confirmation which it affords of the distinction between the initial motor and sensory symptoms as indicative of the seat of the initial discharge in the motor or sensory centre respectively. The cases are those in which a sensory aura descends the limb, and spasm is not associated with it until the sensation reaches the extremity. For instance, a man has attacks which begin thus:—he wakes, in the night, with a 'rushing sensation' in the right shoulder, and this gradually, in about thirty seconds, passes down the arm to the hand, there being no spasm. When it reaches the hand, however, spasm comes on, the hand 'shuts up,' and he loses consciousness. Some cases will be mentioned presently in which the sensory aura, descending a limb, reascends the limb, with the spasm which commences when the sensation reaches the extremity. It seems as if the resistance within the sensory centre is less than between the sensory and motor centres, and that the latter is least in the part in which the extremity of the limb is represented (as may well be when we consider the extremely delicate functional relation between sensation and motion in the fingers and hand). Thus a slight discharge in the sensory centre may extend, gathering strength as it proceeds, until it reaches the part in which the extremity is represented, and then, unable to pass further, it overcomes the resistance between the sensory and motor centres, and sets up spasm in the related muscles, and the discharge passes back in the previously undischarged motor centre, so that, in this case, the sensory aura descends, the motor aura ascends, the limb. But the resistance between the motor and sensory centres, even in the part in which the extremity is represented, must be greater than that within the sensory centre, because in many cases a sensory aura, beginning in the fingers, may ascend the limb without any spasm, as in some cases to be mentioned immediately.

In the majority of cases (three-quarters) in which the attack begins in the hand, consciousness is lost before the seizure has extended beyond the arm. In other cases it passes to the head, trunk, or leg before the patient becomes unconscious. The passage to the head occurred in nine cases, and in four of these it was felt in the side of the mouth. For instance, in one the initial symptom was a sensation of tingling, without movement, in the left hand, followed by similar tingling in the left side of the lips and nose. Occasionally (as in two cases) the aura, passing up the arm to the shoulder, then descends the side of the trunk to the leg and to the foot. These cases will be again alluded to. It is very rare for the aura, after passing to the head, to descend the trunk; but in two cases, after ascending the arm to the mouth, it recommenced in the foot and passed *up* the leg.

Occasionally the side on which the attack begins is not uniform. In one patient the commencement was by a drawing up of one arm, sometimes the left, sometimes the right. A similar variation is occasionally met with in the part of the side first affected, as in a case in which the commencement of the fit was sometimes in the hand, sometimes in the face.

The seizure commenced in the leg in 14 cases, and in ten of these in the foot. In only five did it distinctly begin as a movement, and this began in the toes once only, in the foot as a whole twice. In one case the movement, twitching, began in the hip, and passed down the leg.[1] In five cases the commencement was by a simple sensation, once in the great toe, once in the sole, once in the dorsum, once in the foot as a whole, and once in the leg as a whole. A motor sensation, without movement, was described in one case: the patient suddenly felt as if the leg were bent up under the other, and that he must stretch it out. Of the cases in which the commencement was in the foot, consciousness was lost before the aura had extended beyond the foot in two cases; in six the aura extended up the leg and thigh; in three of these it passed on up the side to the head. In two cases, after passing up the side, instead of going to the head, it passed down the arm to the hand, and then consciousness was lost. In only one case in which it passed up to the head did it afterwards deliberately pass down the arm—just as in the cases in which the aura commenced in the arm, if it ascended to the head, it rarely afterwards passed down the side. The passage of the discharge to the region in which the head is represented appears to be closely connected with loss of consciousness. In two cases the sensation, commencing in the foot, passed up the side, and when it had got to the level of the hand it recommenced there, and then passed up the arm. Conversely, in one case in which the aura commenced in the forefinger, and, after passing up to the other fingers, ascended the inner side of the arm to the face and tongue, while passing up the arm it began in the great toe and passed up the inner side of the leg as far as the knee. There are thus two modes in which the arm is involved secondarily to the leg, just as there are two modes in which the leg is involved secondarily to the arm—one extension by continuity, passing from the one limb to the other by the trunk, and passing down the limb secondarily involved, and the other by separate commencement in the extremity of the second limb, and the passage of the aura *up* both. It seems probable that in the cases in which it passes from one to the other by continuity through the trunk, the discharge in the sensory centre takes the lead, and determines the course of the aura, although it may be closely, even instantly, followed by the motor discharge. The representation in the brain of the cutaneous nerves must be as continuous as the skin in which they are distributed. The passage of the aura by continuity up the arm, down the trunk, and down the leg, or *vice versâ*, is intelligible on this theory, but scarcely on the theory that the discharge in these cases begins in the motor centres.

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[1] A very interesting case of this nature, in which the convulsion commenced in the groin and passed down the leg, was recorded in 1700 by Bonet (*Sepulchretum*, vol. i. sect. 12).

This theory is entirely supported by the details of the cases which have come under my notice. In all cases in which an aura passed from the extremity of one limb to the other limb by the trunk, the warning was sensory in its commencement. The most striking confirmation, however, is afforded by cases (analogous to those in the arm described on p. 48) in which the warning remains purely sensory until the aura has reached the extremity of the *second* limb, and then spasm is superadded. One or two instances of this may be mentioned. In one patient the attacks always began with a sensation in the foot, without motion, and the sensation passed up the leg and side, and down the arm to the middle finger of the hand, and then only was spasm added; the hand began to twitch, and the spasm passed *up* the arm, and then consciousness was lost. In another patient the attacks begin by a sensation of 'numbness' in the left wrist, which passes up the arm, down the side and leg to the foot; then it begins to ascend the limb again, and immediately the limb draws up. In a third case the attacks begin with tingling in the backs of the fingers, and this tingling passes, without spasm, up the arm, and down the side to the leg, and then the leg draws up. A woman has minor attacks which begin with a pain in the toes of the left foot; this pain ascends the leg, without any spasm, passes up the left side of the trunk, to the side of the head, temple, and side of face near the nose, and next passes down the arm and into all the fingers, which feel as if they were swollen; they then begin to twitch; the arm is slightly flexed at the elbow and shoulder joints, and then there is twitching of the face near the eye. When the pain gets into the arm, she becomes unable to move it. There is no loss of consciousness, but she also has severe attacks in which she bites her tongue.

The relation between the motor and sensory phenomena in these cases is no doubt to be explained by the connection between, and the relative resistances in, the motor and sensory centres involved. A discharge beginning in one centre, when it reaches the limit (representation of the periphery), may then, as already stated, not only pass to the related centre, but may return in the same centre, apparently when the discharge is slight, and has little tendency to increase in intensity. In one patient, for instance, minor attacks began with a stabbing pain in the left side of the chest, followed instantly by a 'beating in the inside of the thigh, accompanied by shaking,' which passed down to the foot and great toe, and then returned up the leg, still as 'beat, beat,' to the side of the head, and sometimes it would again return down the side to the toe. In another patient a somewhat similar 'beating sensation began in the big toe and passed up the leg an side of the trunk to the shoulder, down the arm to th fingers, and then back up the arm to the head, when con sciousness was lost.

Associations of unilateral auræ with other warning are comparatively rare. In three cases of attacks com mencing in the arm there was a subsequent special sen aura. In each of these the seizure in the arm began b a sensation proper, not by spasm. This seems to indica that, as might be expected, the special sense-centres a connected with the sensory limb-centres, rather than wi the motor limb-centres. No aura *beginning* in the fac tongue, or leg, was associated with one in the speci senses. The only cases in which the latter was combine with a leg aura were the following. (1) A case in whic the seizures commenced with a pain in the back of th hand, and this pain, as such, passed up the arm and dow the side to the leg. When it reached, still as pain, th middle of the thigh, a flash of light appeared before th eyes. The pain went on down the leg, and when reached the foot consciousness was lost. (2) A patien whose attacks began in the thigh, always suffered fro flashes of red fire before the eyes for some days before th fit; but not at the actual onset of an attack. (3) A patie whose major attacks began with spasm in the left shoulde descending the arm, and followed by spasm in the le orbicularis palpebrarum and left side of the mouth, had als minor attacks, consisting of red and blue lights whirlin before the eyes, and said to appear first before the left ey

In one patient, a girl, a singular association existe The fits began with a feeling of numbness and beating i the right great toe, which passed up the leg, and when reached the groin, the patient felt a need to micturat If she could do so, the aura ceased; if she could not, sh suddenly felt as if turning over and over; then a peculia psychical condition occurred, she lost consciousness, an passed into a severe epileptic fit. I have met with anothe case in which micturition sometimes arrested a commencing fit. I have also met with a case in which the act of micturition sometimes brought on a fit. The shiver, which, in many children and some adults, normally attends the act, indicates the associated affection of higher motor centres, and these facts suggest that the passage of urine during epileptic fits is not merely the result of loss of consciousness, but is really part of the convulsive action.

In only two cases did the attack commence in the side of the trunk; in one the sensation was described as 'burning.' In two cases a left-sided aura was associated with palpitation of the heart.

The mutual relation of the motor and special sense-centres in the cortex of the brain, ascertained by Hitzig and Ferrier, enables us to understand something of the progress of seizures. They do this, whether we regard epilepsy as a cortical disease or not, because Burdon-Sanderson's researches show that the relation is similar in the lower centres. For instance, a fit beginning in the face or tongue, if it involves the limbs, takes the arm before the leg, and we find that the arm centres intervene between those for the face and tongue, near the fissure of Sylvius, and those for the leg, in the upper part of the hemisphere. Again, in a fit beginning with twitching of the angle of the mouth, rotation of the head commonly succeeds, and the centre for rotation of the head is situated in front of, and contiguous to, the centre for the action of the zygomatic muscle. But in other cases it is difficult to understand the progress of the aura from the experimental facts. For this there are probably three reasons:—First, we know little of the degree of structural association or separation of these centres, the cerebral sulci, of course, affording no information on the matter; secondly, the centres represent in many cases complex movements, and fits begin usually with simple movements; thirdly, we are almost ignorant of the mutual relations of the sensory centres for the limbs, which Ferrier's experiments locate on the inner aspect of the temporo-sphenoidal lobe. The facts of many auræ make it probable that these, rather than the motor centres, lead in the discharge, and determine the course of the commencing fit.

A rare but curious condition sometimes attends the onset of a fit which begins unilaterally; it may be termed the alternation of the aura. In some right-sided convulsions, from left meningeal hæmorrhage, which I once watched, the rotation of the head to the right, which accompanied the fit, was preceded by a rotation to the left, and this itself was preceded by a slight initial rotation to the right—a double alternation. I have obtained very few indications of this phenomenon in idiopathic epilepsy. In one case a left-sided fit was preceded by shaking and trembling of the limbs of the right side; in another case the attacks began with a sensation in some left cranial nerve, followed by a sensation around the right wrist.

The great majority of fits which begin unilaterally are purely epileptic. Of 76 in which the character of the fit could be ascertained, only 9 (or 12 per cent.) presented hysteroid convulsion.

*Bilateral and General Warnings.*—In the next group are placed together the bilateral sensations in the limbs, and certain general sensations which seem to the patient to be universal. Of the attacks beginning simultaneously in the limbs of both sides, the commencement was in the arms in eleven cases. In four it was by a sensation only—numbness, tingling, or pain; in seven, by twitching, startling, jerking, or cramp in the limbs, with or without tingling. In most cases the commencement was in the hands as a whole, as might be anticipated from the wide distribution of the discharge. In twelve cases the attacks commenced simultaneously in both legs. In half of these the aura began in the feet, as a tingling, trembling, or creeping sensation. Two of the cases of this group were distinctly due to chronic alcoholism.

Auræ referred to the trunk, and not distinctly visceral, are very rare; and the sensation is almost exclusively felt in the back. It is usually a pain or a sense of trembling in the spine. In one patient the sensation was that of something crawling up the middle of the back, which ascended higher and higher until it reached the back of the head, when there were giddiness, loss of sight, and then loss of consciousness, and an epileptiform convulsion. The spinal aura, not uncommonly, as in this case, seems to ascend to the back of the head. The frequency with which there is hysteroid convulsion in these cases differs very strikingly according to the seat of the aura. All the cases with a spinal aura were epileptic, and so were almost all those in which the attacks began in both arms by a sensory aura or by jerkings or startings, only one of 14 having been hysteroid. But of 11 cases in which the aura was referred to the feet, in only four could I be certain that the attacks were epileptic; in three they were certainly hysteroid, and in four their exact character was doubtful.

Two cases, in which the warning was of this class, afforded the only instances I have met with in which it was of the character which corresponds to the term 'aura.' In one the warning was described as air blowing on the skin of the epigastrium; the sensation ascended to the head, and was followed by beating of the heart and choking. The attack was hysteroid in character. The other case was a lad of 17, whose attacks were apparently epileptiform. The warning commenced with a sensation of guns going off in his head, then his sight became dim, and then he had a feeling as if wind was blowing on the skin, which commenced at both knees, and passed up to the epigastrium, and he then lost consciousness.

The other auræ, grouped together as general, tremor, startings, jerkings, malaise, faintness, were described in 51 cases—one-tenth of the whole. General tremor or shivering, present in 13 cases, was associated in three with pain in the head. General starts or jerkings, the aura in ten cases, were never associated with pain in the head. In several of the cases the fits occurred during sleep; the patient was awakened by the starts or jerks, and then passed into the fit. General sensations were described as preceding the attacks in 15 cases; a feeling of general illness or malaise, of general powerlessness, of general heat (in three cases), of general cold (in one case). A sense of faintness, which the patient could not localise, was the aura in 13 cases, and was associated with sweating in one case and with giddiness in three. Almost all the cases commencing with tremor or starting were purely epileptic; whereas among those which commenced by faintness, one-third, and of those commencing by other general sensations, one-half, were hysteroid.

*Visceral or Pneumogastric Auræ.*—The next group of auræ are those which I have associated as referable to the region of the pneumogastric nerve, including the respiratory part of the spinal accessory. It is not suggested that they have any special relation to this nerve as such. Some of them begin beyond it, as in the lower part of the abdomen;[1] but the pneumogastric is the nerve to the organs in which visceral auræ are most frequent, and the majority of these auræ are confined to its functions. These warnings were described in 106 cases.

The commonest form is the so-called 'epigastric aura,' a sensation referred to the pit of the stomach, present in one-half (51 cases). In a third of these it was actual pain; in half it was a vague indescribable sensation; in the remainder it was described as a feeling of 'burning,' of 'coldness,' of 'trembling,' of 'something there,' or of 'winding up.' In most cases it was referred to the epigastric region generally; in a few it was a little to the left; in none to the right. In two it commenced lower in the abdomen; in several it was referred to both the epigastrium and the lower part of the sternum. Whenever the sensation was actual pain, it remained at the epigastrium until consciousness was lost. In many of the cases in which the sensation was vague, it was described as 'rising up through the chest' to the throat or head. The passage to the head was usually followed by immediate loss of consciousness; that to the throat by a sensation of choking. This latter reminds us of the well-known sensation in hysteria, and in many cases of genuine epilepsy the sensation appears to be exactly the same as the globus hystericus.[1] Not only so, but I have seen a case of tumour of the pons Varolii in which epileptiform convulsions commenced with the same sensation, described as a 'ball rising up from the stomach to the throat, and stopping the breath.' The sense of choking is sometimes very intense. One patient described it as if the palate and larynx were pressed together.

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[1] It must be remembered, however, that the pneumogastric also innervates the intestines.

Just as the warning commences sometimes to the left of the epigastrium, but never to the right, so when it is felt as ascending through the chest on one side, this is always on the left side, never on the right.

In 18 cases in which there was no sensation at the epigastrium; the warning was the choking feeling only. In two of these there was also coughing. In one patient, left-sided fits always commenced with attacks of retching and spitting, but not vomiting. In seven cases the attacks commenced by some other sensation in the throat. One patient would suddenly exclaim that her throat was sore, grasp it with her hands, and fall in the fit. In another case the warning was 'tingling' in the throat. In six cases there was a sensation of dyspnœa or suffocation, referred to the chest and not to the throat. In five cases there was some other sensation in the chest, vague in two, 'burning' in three.

These auræ felt in or ascending to the chest and throat appear to be the expression of a disturbance in the sensori-motor processes connected with the respiratory function of the pneumogastric. I need hardly point out that spasmodic actions form part of the normal action of the respiratory centre as they do of no other, and must be reproduced in higher and associated centres. The epigastric pain which remains limited to the epigastrium appears to be related exclusively to the gastric division of the vagus. Accordingly we find that in 14 cases there was described, as part of the aura, another sensation referable to the same function—nausea, evidently such after most careful questioning, and accompanied in several cases by actual retching. In one case sudden, intense hunger was described. Other symptoms, often associated with gastric disturbance, accompanied the pneumogastric aura in some cases, as pain in the head and giddiness, the latter especially when there was actual nausea, the former when there was actual pain in the epigastrium.

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[1] The close association of the globus hystericus and epilepsy attracted the attention of Dr. Parry. (*Medical Writings*, London, 1825, p. 392.)

Another organ within the innervation of the vagus is the heart, and cardiac sensations constituted the aura in 16 cases, pain in two, a vague sensation in three, and palpitation in eleven. In one patient the attacks were usually preceded by an epigastric sensation passing up the left side of the chest to the head, but in some attacks this was preceded and followed by severe palpitation of the heart and dyspnœa. Cardiac palpitation and discomfort are sometimes associated in an aura as 'tingling' in one or both arms.

Hysteroid attacks occurred in a larger proportion of the cases in this than in any other group—one-fourth. They constituted one-fifth of those with an epigastric aura, or with chest dyspnœa, one-half of those with a throat aura, and one-third of those with a cardiac aura.

*Vertigo.*—The next group comprehends the cases, 90 in number, in which the attacks commenced with a sensation of giddiness, vague or definitely vertiginous, or with the fact of rotation. As a rule, when a sensation merely, the direction could not be ascertained, but of five cases the sensation was of turning to the right in two, and to the left in three. Of 15 cases with actual rotation, the movement was to the right in five, and to the left in ten. In nearly all the convulsion was bilateral. In bilateral fits the discharge is never quite equal on the two sides, as proved by the common deviation of the head, and the vertigo, when slight, may be the expression of this inequality (see p. 73). Almost all the cases were epileptic, one-sixth only presenting hysteroid convulsion. In most consciousness was very early lost.

As already stated, giddiness is often associated with an epigastric sensation, especially when this is distinct nausea. It may also be associated with loss of sight. In one patient, for instance, objects before him suddenly appeared to turn round and round, and, while doing so, gradually became indistinct, and he lost consciousness.

*Cephalic Auræ.*—Sensations referred to the head preceded the fits in 50 cases. In 21 the sensation was actual pain, mostly general or undefined, in a few frontal, in very few vertical or unilateral. In one patient the warning was violent pain through each zygoma, and after the fit there was local tenderness at the seat of the pain. These cases are important, because, when the attacks are slight, they may be regarded as simply neuralgic. Cephalic pain is rarely associated with any other aura. In 29 cases the sensation was other than pain, usually vague, in a few definite—described as 'blood rushing to the head,' 'coldness,' 'burning,' or a dim sense of heaviness or pressure. In some cases it was associated with distinct vertigo, and in others the cephalic sensation itself was closely allied to vertigo. In one patient, for instance, the sensation was always described as 'two circles within the head moving rapidly in opposite directions, and seeming to get alternately nearer and farther off,' until consciousness was lost. In one or two cases a cephalic sensation was associated with an auditory aura, in none with a visual sensation. In one case it preceded spasm in the arm—the patient felt 'as if his brains were being stirred up by some instrument,' and then the arm was drawn up with the fingers spread out. In one-fourth of the cases with this aura the convulsions were hysteroid, and the proportion was nearly the same whether it was a mere sensation or actual pain.

Sudden somnolence preceded the fit in five cases, all purely epileptic. Sudden inability to speak constituted the warning in nine cases, of which two were hysteroid.

*Psychical Auræ.*—The aura was psychical in 25 cases, an emotion in 10, an idea in 15. The emotional auræ in all took the form of fear—vague alarm or intense terror. Associations with other auræ were rare, the only combinations being with a sensation in the legs and with pain or other feelings in the head. Hughlings Jackson has drawn attention to the occasional association of this aura with an epigastric sensation, but this association is extremely rare. I have only twice met with it: (1) In the case of a girl aged 13, all of whose fits began with a sensation of terror, and she would sometimes run across the room with a look of great alarm before an attack; at other times put her back against a wall with an expression of fright, much to the astonishment of her friends. This constituted the whole of the slight attacks, but she also had severe epileptiform convulsions. During four years the feeling of fright constituted the sole warning; then she had one attack in which this was associated with an epigastric sensation. (2) In the other case the slight attacks were marked by a manifestation of extreme terror which the patient did not afterwards remember, and this was preceded by a vision of red stars or by an epigastric sensation. I have enquired for the association in a large number of cases with an epigastric aura, but with these two exceptions I have always failed to obtain a history of it.

In every case with an emotional aura the fits were purely epileptic, although in some they were slight. This is remarkable, since, as we shall see, manifestations of terror are common in hysteroid attacks. The occurrence of this aura bore no relation to fright as a cause of the disease. Its comparative rarity may excite surprise. No case was reckoned in which the sensation was not mentioned spontaneously by the patient. In most, perhaps in all, cases with a deliberate onset, a natural feeling of alarm is produced by the warning, and if the patient is asked if he has a feeling of fear, he usually replies in the affirmative;

but this can evidently not be reckoned as a true emotional aura.

Of the 15 cases in which the attacks were preceded by a mental state, in 12 they were purely epileptic, in three hysteroid symptoms occurred. In most cases no other description of the aura could be given than that 'an idea seems to come into the mind.' In one case, that just mentioned as commencing by a sensation in the great toe, followed by a desire to micturate and a feeling of turning over and over, this was succeeded by the idea of being in heaven, seeing persons and hearing a question asked. The subsequent fit was certainly epileptic. Several patients described a feeling analogous to that which Hughlings Jackson has designated 'voluminous,' a 'dreamy state,' sometimes apparently similar to that said to be experienced during the act of drowning, a sudden recollection of many past events.[1] Another occasional aura was a sense of strangeness—familiar objects suddenly seemed unfamiliar, and then the attack came on. In one case whatever was taking place before the patient would suddenly seem to be wrong—*i.e.* morally wrong—and then he lost consciousness.

*Special Sense Auræ.*—The last group of auræ are those referred to the special senses and their organs, which were present in 119 cases. Two-thirds of these (84) were referred to the organ or sense of sight, one-fifth (26) were auditory, nine olfactory or nasal, and one gustatory.

*Olfactory.*—Sensations of smell preceded the fit in seven cases, all purely epileptic. In most cases the sensation was of an unpleasant smell. One patient described it as a smell 'like matter;' another as a smell like sulphur which seemed to 'stir up from the pit of the stomach;' another such as is caused by burning dead bodies; another as like rotten eggs; another compared it to Tonquin beans. The only associations noted were with a visual aura (two cases) and with a feeling of suffocation (two cases). The nasal warning was present in a few cases, all epileptic, and consisted in sudden tingling in the nose.

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[1] An interesting discussion of these mental states in relation to epilepsy is given by Dr. Hughlings Jackson in his 'Lectures on the Diagnosis of Epilepsy,' *Brit. Med. Journal*, February, 1879, p. 141.

*Gustatory.*—A true gustatory aura is among the rarest forms of warning, and was met with in only one case. It was described as a taste between sour and bitter, and was referred to the back of the tongue. A gustatory aura occurred, however, in another patient with post-hemiplegic convulsion—a metallic taste in the mouth like copper, with a simultaneous sensation in the thumb and forefinger of the left hand, as if they were being pinched with a hot iron; the sensation then passed to the other fingers, and then up the arm to the side of the face and down the body to the leg, and 'seemed to go out at the great toe.'

*Ocular and Visual.*—The warnings referred to the organ of vision may be divided into five classes: a sensation in the eyeball itself; diplopia; an apparent increase or diminution in the size of objects; loss of sight; distinct visual sensations. A sensation in the eyeball was present in seven cases, five purely epileptic. The eye to which it was referred was in one case on the same side as the first motor symptom of the fit; in one on the opposite side. It is probable that the centre for common sensation in the eyeball is connected with that for vision. This is suggested by the phenomena of photophobia due to the fifth nerve. In one patient, fits commencing with a pain in the eye were followed by red vision; and in a case of cerebral tumour, presently to be mentioned, a visual sensation and pain in one eye were associated as the aura of the fits. Double vision preceded the attacks in five cases. Two were cases of hysteroid attacks, and the diplopia may have been due to the convergent strabismus so common in hysteroid states. In two of the cases, however, the attacks were epileptic. In one the diplopia was associated with aching in one eye and an apparent magnification of objects, which looked 'twice their real size.'

The apparent increase or diminution in the size of objects is a rare and curious aura. It is apparently connected with increase or diminution in the sensitiveness of the visual centre, which produces the same effect on the consciousness as the stimulation of a larger or smaller area of the retina by a larger or smaller image. It is therefore not surprising to find that diminution may be followed by loss of sight. For instance, in one case objects appeared to recede, becoming smaller and less distinct. If the patient was going to a door, for instance, she would have to feel for it, because it appeared small and indistinct; the indistinctness increased until sight was almost gone, and then consciousness was lost. Loss of sight preceded loss of consciousness in 26 cases—18 purely epileptic, four hysteroid. It was commonly complete and simultaneous in the two eyes. In one case the sight of the right eye went before that of the left, and this was followed by rotation of the head to the left. In one case the loss of sight in minor attacks lasted for an hour, and was followed by sleep for an hour. The loss was preceded by a visual sensation in two cases, by an auditory sensation in one, and in one case loss of hearing followed the loss of sight. The only frequent associations were with vertigo in four cases, and with rotation of the head in two.

The aura was a distinct visual sensation in no less than 46 cases (35 epileptic, 3 with hysteroid symptoms, 8 doubtful). The sensation in nine cases was that of light, not colour—sparks, a ball of light, a flash, or a glare. In 17, colours were seen; and 15 patients were able to give some account of the colours. In seven, only one colour was seen, and this was always either red or blue—the former described five times, the latter twice. No other colour was ever seen alone. Combinations described were —red and blue twice; red, blue, and purple once; red, blue, and green once; red and green once; blue, green, and yellow twice; red, blue, and yellow once. Thus not only were red and blue the only colours seen alone, but they were both present in half the cases in which other colours were seen, and in no case were both absent. Taking the cases together, the order of frequency of the several colours is—red (eleven times), blue (eight times), green (three times), yellow (three times), and purple (once). It is evident that this order does not correspond to the physical relations of colour, as shown, for instance, by their grouping in the spectrum; nor does it correspond in any way to the areas of retinal perception. Red, seen most frequently, has one of the smallest retinal fields; but green, seen much less frequently, has a still smaller field; while the field for yellow, seen as rarely as green, is one of the largest, and is nearly the same as the field for blue, which is one of the colours mentioned most frequently. But the order does correspond, not exactly, but in a very suggestive manner, to the degree of visibility of colours. Cohn[1] found that the order in which spots of colour, one millimètre in diameter, can be seen in direct sunlight at different distances, is—beginning with that which can be recognised at the greatest distance—red, next blue and green (equal), next yellow, and least violet. This is evidently not far from the order of development in epilepsy.

The apparent diminution in the size of objects, which must be referred to an inhibition of the visual centre, has been mentioned in connection with the loss of sight which it often precedes, but must be again alluded to in connection with these visual sensations. Partial inhibition is equivalent to a diminution in the sensitiveness of the centre. An object appears smaller and farther off, and as the inhibition of the centre increases, the object may appear to recede. It is the opposite process to that which occurs in discharge of the centre; in this there is increased sensitiveness, and objects appear larger. But a discharge commonly results in such liberation of energy as affects consciousness. There is a visual illusion, perhaps a sense of a ball of fire; as the discharge spreads this seems to get larger and nearer. The effect is the same as if an increasingly large area of the retina were stimulated. Thus in one case the warning was always a blue star, which appeared to be opposite the left eye, and to come nearer until consciousness was lost. Another patient always saw an object, not described as light, before the left eye, whirling round and round. It seemed to come nearer and nearer, describing larger circles as it approached, until consciousness was lost. The phenomena of some of these auræ suggest that there may be both a discharge and inhibition of the same centre. For instance, one patient always first saw a flash of light, then sight was lost, and then consciousness. So the light or other object which appears to the patient, instead of becoming larger, may seem smaller and more distant, apparently in consequence of the inhibition overcoming the discharge. Thus in one case the warning commenced by dimness of sight, and then a light appeared, whirling round and round. This appeared to get farther and farther off, until consciousness was lost. There may be inhibition of the centre in severe attacks, and a discharge in the minor seizures. Thus one patient always saw colours, especially green and blue, in slight attacks, while the severe fits were preceded by simple loss of sight.

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[1] *Brit. Med. Journal,* Oct. 4, 1879.

In fourteen cases the aura was a highly specialised visual sensation, a visual idea. In two cases this succeeded a simple visual sensation. One patient, in attacks of *petit mal*, first saw a number of red and white dazzling stars, and then an old woman, and other objects which she could not remember. This is of course rather an attack than an aura. But another patient first saw always bright lights—red, green, yellow—and then, the lights continuing, she saw a girl, and 'tried to get to her to ask what the lights meant,' and then fell in the fit. In the first of these cases the less specialised preceded the more specialised sensation, a sequence to which Hughlings Jackson has drawn attention, as suggesting that the more elaborate sensation is the result of the loss of control from the discharge causing the less elaborate sensation. But in the second the more and the less elaborate sensation occurred together, and in other cases an equally specia sensation, a visual idea, constituted the sole warning of the fit, and must be ascribed to the discharge itself. Tw patients had visions of an old woman; in one case, to b mentioned in detail presently, the old woman always had a brown dress—a form of aura of which several instances hav been recorded—and in three ugly faces were seen, and i two 'various people;' in one 'animals.' Two patient described an aura consisting of visions of objects and place familiar long ago, a visual sensation analogous to th psychical aura I have mentioned. In one patient the fi were preceded by a vision of 'beautiful places, larg rooms,' &c.[1]

Associations of the visual aura are comparatively rar In three cases the conjoined aura was in the limbs, and, a already mentioned, in all these the aura in the limb com menced by a sensation. One of these was the case in whic a flash was seen as soon as a pain, which commenced in the arm, reached the middle of the thigh. When the visua sensation is unilateral, the side may be the same as that on which the limbs are involved. Thus, in one case, some fits commenced with the appearance of a blue star before the left eye, which grew larger and larger until consciousness was lost, and other fits began in the left arm, and others in the left leg. In another patient a bright light referred to the right eye was associated with loss of speech. In these cases the sensation may have been rather referable to one side of both fields of vision than to one eye. Occasionally a visual aura is associated with initial rotation, of which the patient is aware. Thus, in one, a blue star always appeared and seemed to move to the left, and the patient felt obliged to follow it with his head: the subsequent fit was apparently bilateral. Epigastric sensations were associated in three cases, and in three an auditory sensation. The sensations in one patient were very complex and numerous, and, as I shall have to refer to it again, it may be well to relate the case in some detail. The patient was an intelligent man, twenty-six years of age, and all his attacks began in the same manner. First there was a sensation in the left hypochondriac region 'like pain with a cramp;' then, this sensation continuing, a kind of lump seemed to pass up the left side of the chest, with a 'thump, thump,' and when it reached the upper part of the chest it became a 'knocking,' which was heard as well as felt. The sensation rose up to the left ear, and then was like the 'hissing of a railway engine,' and this seemed to 'work over his head.' Then he suddenly and invariably saw before him an old woman in a brown-stuff dress, who offered him something which had the smell of Tonquin beans. The old woman then disappeared, and two great lights came before him—round lights, side by side, which got nearer and nearer with a jerking motion. When the lights appeared the hissing noise ceased, and he felt a choking sensation in the throat, and lost consciousness in the fit, which, from the description, was undoubtedly epileptic. He had also attacks of *petit mal*, which consisted of a vision of a dull-red ball to the right, in the lower part of the field. Thus here we have first a pneumogastric (visceral) sensation which becomes intermitting and sound-like as it ascends towards the ear, until it is finally an intermitting hissing, perhaps the least specialised of all the auditory sensations. This is accompanied by a visual sensation of very high specialisation—a visual idea—associated with an olfactory sensation, and it gives place to a much less elaborate sensation, the two lights, and, as the visual centre becomes invaded by this discharge, that in the auditory centre ceases. Finally, the aura terminates with the completion of the pneumogastric sensation—a sense of choking. The case will be referred to again in the chapter on 'Pathology,' but I may here point out that the more specialised visual sensation preceded the less special, and therefore cannot have been due to 'loss of control' by preceding discharge; it was evidently due to the discharge itself. The phenomenon of *two* lights before the eyes is one of great rarity among the auræ of epilepsy. Lastly, the association of intermission of the sound with a jerking movement of the lights is worth remark. In one other patient a visual aura was accompanied by a disagreeable undescribable 'smell,' after it had lasted a short time, and just before consciousness was lost.

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[1] Cases of slight epileptic attacks with a highly specialised visual aura have been lately described by Hammond as 'thalamic epilepsy' (*Neurological Contributions*, part iii.); but the evidence that they depend on a morbid process in the optic thalamus is at present too slender to justify the name.

The only autopsy which I believe has been published of a case in which the visual warning existed, is one which I recorded two years ago.[1] The fits were preceded by a flash of light or pain in the eye, and micropsy, and were due to a tumour in the occipital lobe, which had extended forwards as far as the visual centre in the angular convolution.

*Auditory.*—Various forms of auditory warnings were noted in 26 cases. In six there was an auditory loss, a sudden sense of stillness, analogous to the sudden loss of sight already mentioned. In one case the two were associated; for a moment all was silent, then all was dark, then consciousness was lost. In a case in which the sensation began in the tongue and passed to the arm and leg, sight was lost after the arm was involved, and hearing after the sensation was felt in the leg.

Auditory sensations preceded the fits in 21 cases. In four it was a sensation of an explosion or crash, an aura noted by Aretæus; in other cases the sensations described were the 'sound of a drum' (twice), a 'whiz' (twice), a 'hissing' (twice), a 'ringing' (twice), a 'rustling,' a 'rushing sound,' and a 'sound of thunder,' or a 'whistle.' In some cases the sound was at first distant, and seemed to get louder and near, until consciousness was lost—a phenomenon analogous to that just described in connection with the visual aura. In one case some attacks were preceded by an auditory sensation, 'singing in the ears,' and others by an auditory loss,—sounds suddenly became distant and indistinct. All these may be regarded as the lowest forms of auditory sensation. In three cases the sensation was more special—an auditory idea. One patient always heard the words 'c-o-n with a con' repeated several times, and then came the fit. In two cases music was heard. One of these cases illustrates the fact that the slighter the discharge, the more elaborate is the sensation. Slight fits were preceded by the sound of music, a confused 'air,' in which the patient could never succeed in making out the tune. Before severe attacks, however, he heard only a bell ringing.

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[1] 'Cases of Cerebral Tumour illustrating Diagnosis and Localisation,' *Lancet*, 1879, vol. i.

The most frequent associations of the auditory aura were with a visual aura (in seven cases), and an olfactory aura (in two cases). In two cases it was associated with a sensation beginning in the left hand, and other combinations noted were with auræ beginning in both legs, in both arms, with vertigo, an epigastric sensation, and nausea. In the three latter we may trace the normal association of the auditory centre. In several cases in which a visual and auditory aura were combined, the two corresponded in character—*i.e.* in their degree of elaboration. For instance, one patient described a red light and a rushing sensation like water. The patient who heard the words 'c-o-n with a con,' saw a girl standing by her who was saying them; and a patient who saw 'beautiful places, large rooms, &c.,' heard at the same time 'beautiful music.' An auditory sensation may, however, be accompanied by dimness of sight, as in the case in which these symptoms were accompanied by a sensation of a wind blowing upon the skin, mentioned at p. 55.

The association of the visual and auditory warnings is well explained by the contiguity of their centres as determined by Ferrier. The angular gyrus, in which is the visual centre, is continuous with the superior temporo-sphenoidal convolution, in which is the auditory centre.

I am aware of no published post-mortem of a case in which an auditory aura preceded the fits, but in a case of Dr. Hughlings Jackson's, of cerebral tumour (which I had the opportunity of watching with him for some years, and which he kindly permits me to mention) a sound of bells ringing, referred to the left ear, preceded left-sided fits in an early period of the disease. After death we found a tumour outside the opposite optic thalamus. It was extensive, but had evidently commenced between the thalamus and the upper temporo-sphenoidal convolution.

Before leaving the subject of modes of onset, one other point may be considered—the relation of the side on which the attack begins to its characters. Hughlings Jackson has suggested that the senses are not equally represented on the two sides of the brain, and that the more special the sense, the more unequal is its representation, the more does it preponderate on the right side. The facts of this series of cases give only a partial support to this view. We have seen that attacks commencing by a sensation in the arm begin twice as frequently in the left arm as in the right. Nevertheless, all the cases commencing by a sensation in the tongue were right-sided. In none of the cases with an olfactory aura was there any indication of the side which 'led' in the fit. In most of the cases with an auditory aura, the sensation was referred to both ears. In six cases, however, it was referred to one ear, twice to the right (left side of brain), and four times to the left (right side of brain). But in ten cases in which the commencement of the fit was attended by a visual sensation, and in which there were indications of the leading side, it commenced on each side in the same number of cases (five), and we must regard the visual as the most special of all the senses.

## CHAPTER III.

### *SYMPTOMS CONTINUED.*

### *SEVERE ATTACKS.*

THE symptoms of the developed attack, briefly described on p. 36, may now be considered in detail.

*Epileptic Cry.*—The onset of some attacks is attended by a well-known scream, the 'epileptic cry.' It is more frequently absent than present. Its characters vary. The true epileptic cry is a wild, harsh, screaming sound, probably due to the tonic spasm of the thoracic and abdominal muscles expelling air through the glottis, narrowed by spasm. The sound has been compared by Reynolds to the 'cry of a distracted peacock.' It is indeed scarcely imitable except by the larynx of a bird. There is a parrot at the country branch of the Hospital for Epilepsy which has learned to imitate the cry so closely as frequently to deceive the nurses, who run to the spot, expecting to find a patient in a fit. In many cases the cry is not of this character, but is a simple scream, sometimes repeated once or twice, as the fit is coming on. No recollection of the cry is, as a rule, retained. In rare cases the patient is conscious of the scream, although he is unable to prevent it. Reynolds has recorded an example of this, and I have met with one or two instances.

*Loss of consciousness* is invariable in all severe epileptic fits. It is only in minor seizures that, in rare cases, some consciousness may be retained. In many instances the loss of consciousness occurs at the very onset of the fit, before, or at the same time as, the earliest convulsive symptom, so that the patient knows nothing of the onset. In other cases, as already detailed, consciousness may be retained until after some other symptom has occurred, of which the patient is conscious, as the warning of the fit, and, in the description of these warnings, full information as to the period at which loss of consciousness occurs has already been given. It is always lost before the patient falls. The loss usually occurs later in cases of convulsion from organic brain disease than in idiopathic epilepsy.

CONVULSION. *Tonic Spasm.*—As a rule, the convulsion of a severe epileptic fit begins by tonic spasm, causing violent, strained distortion of head, face, and limbs.

The first evidence of the commencing spasm is, in a large number of cases, a deviation of the head and eyes to one side, or an actual rotation of the head. Sometimes the head is turned 'as far as it will go,' without any rotation of the trunk. Occasionally the trunk is rotated, and the patient may even turn round, once, twice, or three times, before falling. The eyes are always directed towards the side to which the patient turns. The mere deviation of the head is probably the result of the discharge being greater on one side than on the other. This is shown in certain severe fits in which a transient post-epileptic phenomenon (foot-clonus) succeeds the fit. If there is initial rotation of the head, the foot-clonus may be found only on the side towards which the head had been rotated. It is shown also by the phenomena of fits which are unilateral or begin unilaterally; the head and eyes are first turned towards the side on which the convulsion commences. The head is turned by the opposite sterno-mastoid. Each of these muscles acts in physiological association with the muscles of the limbs on the opposite side, turning the head, for instance, towards the arm which is in use.[1] The association is of the movements, not necessarily of the muscles. In a right-sided epileptic fit the spasm involves the *left* sterno-mastoid, turning the head to the right. The lateral muscles of the eyeballs, acting together, turn both eyes towards the side affected. If the spasm, after affecting the limbs on one side, passes over to the limbs on the other, lessening on the side first affected, the head and eyes are turned towards the second side when this is involved. The condition in unilateral convulsion (as Hughlings Jackson has pointed out) is always the opposite to that in unilateral paralysis. In the early stage of hemiplegia the head and eyes deviate *from* the paralysed side in consequence of the weakness of the muscles which should turn them to that side. They thus deviate towards the side of the cerebral lesion. In the unilateral convulsion there is deviation *from* the side of the brain affected, and *towards* the limbs which are convulsed.

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[1] This physiological association determines the pathological associations of the action of the muscles; as, for example, in torticollis. In cases of that disease in which the spasm involves first the sterno-mastoid, and afterwards one arm, it is always the arm on the opposite side which is involved.

The alternation of the initial rotation of the head, mentioned on p. 54, is a very rare and remarkable phenomenon. A symptom which may belong to the same category is presented by a patient now under treatment for local convulsive attacks, affecting the muscles of the face, of mastication, of the tongue and palate on both sides, lasting about half a minute, without loss of consciousness. About a minute before each attack there is a sudden deviation of the head and eyes to the left; they are fixed for a few seconds in tonic spasm, which then passes away. A minute later the fit comes on.

To return to the ordinary epileptic attacks. When there is not merely deviation but an actual extreme rotation of the head or a rotation of the trunk, there is probably not merely an excessive action of the motor centres on one side, but of the centres for rotation. The evidence of this is that in purely unilateral fits there is not necessarily, or even usually, any actual rotation, and in the cases in which this does occur the attacks are commonly severe and bilateral. But although actual rotation does not necessarily occur in unilateral fits (and so is not a simple consequence of unilateral discharge), it may occur at the commencement of fits which begin unilaterally, just as at the commencement of bilateral fits. In a patient, one of whose attacks I witnessed, the first event was that her head and eyes were suddenly turned to the right, then she turned round one and a half times, and would apparently have gone on turning had she not fallen; as she fell the spasm began in the right side, and afterwards involved the left.

The facial muscles pass into tonic contraction, greatest on the side towards which the head deviates. It is usually most marked in the zygomatici, and hence the mouth i usually twisted towards one side. The orbicularis palpebrarum is sometimes little affected, sometimes conside ably, and, in the latter case, more on the side toward which the head turns than on the other.

The posture assumed by the limbs under the influenc of the tonic spasm varies in different cases. The mos common posture of the arms is slight abduction at th shoulder-joint, flexion of the elbow-joint to about a righ angle, very strong flexion of the wrists, while the finge are flexed at the metacarpo-phalangeal joints, and extende at the others, the thumb being adducted into the palm pressed against the first finger. The degree of flexi thus increases towards the periphery, but the fingers a in the position, not of simple flexion, but of interosse flexion, *i.e.* that produced by action of the interossei- flexion at the metacarpo-phalangeal articulations, exte sion at the others, with the thumb pressed against the fir finger or beneath it. The position, so far as the digits a concerned, is nearly that of grasping a pen. The san posture is seen in many cases of athetosis and of paraly agitans—a significant fact, indicating that the san nervous processes, which are in overaction or unbalanc action in those diseases, determine the form of the spas in many cases of convulsion. The legs are common extended, the feet inverted. The extension may not complete; there may be slight flexion at the hip and kn joints. Sometimes initial extension of the legs gives place to strong flexion in the later stages of the fit. The spasm may be strictly symmetrical on the two sides, but more commonly there are slight differences in the degree of flexion of the arms, or of extension of the legs.

It may be noted that in this spasm we have reproduced the form of muscular action which is, physiologically, the most frequent and the most important. The chief use of the arms is in flexion, of the legs in extension. The nervous arrangements for these movements, respectively, will thus be the more developed. Moreover, the movements of the arms increase in complexity, and so in the development of their nervous arrangements, towards the extremity. Hence the flexor spasm increases towards the extremity. Further, all the more important and delicate actions (as writing, &c.) in which the fingers are concerned are those in which the interossei take the leading part. Strong flexion of the second and third phalanges is chiefly employed in comparatively coarse movements, in providing, for example, an instrument by which the larger muscles of the shoulder and upper arm, &c., may do energetic work, as in lifting a weight. Hence we find that in the most typical forms of convulsion the interosseal position is assumed in the spasm. In another class of fits, however, especially those in which strong extensor spasm predominates, the long flexors of the fingers are chiefly involved, and the 'fists are clenched.'

Occasionally, instead of this combination of flexor and extensor spasm, flexion predominates throughout. The head is bent forwards, the arms are strongly flexed at the elbow-joints, and the legs at both hip and knee joints, so that the knees are drawn up to the abdomen. In some of these cases also the fingers are strongly flexed at all joints by the long flexors. In other cases, again, the spasm in the arms is strong flexion, the clenched fists being brought up against the chest, while the legs are in extensor spasm. These flexor fits often begin by falling forwards.

It may be noted that patients, before losing consciousness, sometimes have a sensation of this flexor convulsion, although none exists. I have only met with this in cases of nocturnal epilepsy. One patient, for instance, always woke up from his sleep with the sensation of being 'twisted up into a ball with his legs round his neck,' and then lost consciousness in a severe epileptic fit. All the time his limbs were extended and rigid. Another patient also wakes up suddenly in the night, and immediately has a sensation as if his arms and legs 'were all twisted up together,' and then loses consciousness. His limbs are always straight out, and rigid.

In rare cases the arms are raised up above the head at the onset of an attack, and may be kept in that position through a distinct epileptic fit. Or, after being raised, they may be put straight forwards. In some patients, in whom the arms are held up throughout the attack, one or both legs are strongly flexed. In other cases of this kind the legs are extended and rigid. The raised arms may be flexed at the elbow-joint. In these attacks the head is often bent back.

In other fits, again, there is no flexion of the elbow-joints: the arms are straight out and rigid. In these the fingers, as already mentioned, are often strongly flexed at all the joints, but rarely, if ever, are in interosseal flexion.

The bilateral muscles of the body are involved in the tonic spasm as well as the limbs. The jaw is fixed and may deviate to one side, and the muscles of the thorax and abdomen also become rigid.

The violence of the tonic spasm is often very great; the shoulder may be dislocated by it; and in patients in whom this accident has once happened, it is apt to occur again in subsequent fits. In a case recorded by Trousseau the occurrence of fits was not known until it was found that the patient's shoulder had been dislocated during sleep. Dislocation of the lower jaw is said sometimes to be produced, but it is extremely rare.

*Clonic Spasm.*—After the tonic spasm has lasted for a few seconds or for one, two, or three minutes, the stage of clonic spasm comes on. Its onset is not sudden but gradual. In the most typical cases the tonic spasm becomes distinctly vibratory in character; the vibrations become less frequent and more distinct, until they consist of a series of clonic contractions. The remissions, as they become longer, become greater in degree, until at last they become complete intermissions, and towards the end of the fit the limbs are completely relaxed between the successive shock-like jerks. As the remissions become greater the contractions apparently become stronger, but the increase in strength is apparent only, and due to the fact that each contraction starts from a greater degree of relaxation. The spasm ends by the remissions becoming longer and longer until, when they amount to one or two seconds, the jerks cease; but the last contraction is often as strong as any which have preceded it.

The muscles involved in the clonic spasm are usually the same as those which were involved in the tonic spasm. As the clonic stage developes, the limbs retain the same position in which they were fixed by the tonic spasm, commonly with the arms in flexion and the legs in extension. During the intermissions the limbs may fall from the position they occupied before, but each clonic jerk tends to bring them back to it. In the most frequent posture, for instance, in each jerk the elbows are flexed, and the wrists flexed still more strongly, and the fingers jerked at the metacarpo-phalangeal joints.

The clonic spasm involves the muscles of the face, and also those of mastication, of the tongue, soft palate, and larynx. I have twice watched the soft palate jerking up and down synchronously with the other muscles. It is usually during this stage that the tongue is bitten; the spasm in the muscles of the tongue pushes it between the teeth while the jaws are being jerked by the spasm in the masseter and temporal muscles. The tongue may, however (as Trousseau pointed out), be bitten during the stage of tonic spasm, being caught between the teeth as the rigidity comes on. It may thus be bitten in fits in which there is only tonic spasm. The clonic spasm affecting the muscles of the thorax causes the air to be forced intermittently through the lips, and frothy, often blood-stained, saliva is then ejected, constituting what is termed the 'foaming' at the mouth. The blood often comes from the bitten tongue, but sometimes seems to proceed from the mucous membrane during the vascular turgescence.

Another form of clonic spasm is sometimes seen in epileptic fits. Instead of the shock-like clonic spasm, into which the tonic spasm gradually passes, the spasm consists of finer movements, of greater rapidity and less range, and is superadded to, instead of superseding, the tonic spasm. The latter continues, and the strained rigid limbs present this fine rapid movement, almost like a coarse tremor, which maintains the same rapidity instead of becoming less frequent. This form of spasm is often seen in the cases which present flexion of the fingers at the joints and tonic extension of the limbs. Sometimes it occurs in cases with flexor tonic spasm. Very rarely the two forms of clonic spasm are combined in different limbs, as in a case described on p. 86.

Although most attacks of epilepsy consist of both tonic and clonic spasm, in some fits there is but one form of spasm, tonic or clonic. As a rule, those which consist only of tonic spasm are general fits of slight severity. A patient falls unconscious, is rigid for a few moments, and then is better. Occasionally more severe attacks consist only of tonic spasm, as the case of a boy of whose fits the following is a description. His head was first turned to the right; then his arms became extended and rigid, the right being more abducted from the body than the left. Both elbow-joints were then flexed, and his fingers were flexed in the interosseal position. In a few moments the spasm ceased, lasting a little longer in the hands than elsewhere. There was no clonic spasm. He had also more severe attacks, consisting only of tonic spasm, but in them the arms were raised above the head; there was foaming at the mouth; and the tongue was bitten. These severe tonic fits (the 'tetanoid epilepsy' of Pritchard) are not common.

In some patients who present both forms of spasm, the tonic stage is very long, and the clonic stage is very short. For instance, a boy, in one attack which was witnessed, put his head a little back, his arms were put forward, and he became rigid. The trunk was twisted a little to the right, and the face was drawn towards the right side. This continued for about thirty seconds; there was then slight general clonic spasm for two or three seconds, and the attack was over.

Cases of severe convulsion, general from the first, in which the spasm is entirely or almost entirely clonic, are not very common, but are occasionally met with. Some of them are related to other forms of convulsion, especially to hysteroid fits. I once witnessed a striking example of this form of convulsion in a girl aged eighteen, who had also aortic regurgitation, with much hypertrophy of the left ventricle. She was distinctly hysterical, suffered from other pure hysteroid attacks, from frequent clavus and globus hystericus, and also from an attack of rhythmical hysterical chorea, which came on when another patient was admitted to the ward suffering from the same affection. But the attack I am about to describe, although differing from ordinary epileptic fits in several features, yet resembled them, in many important characters, most closely. She was sitting in a chair by me, answering questions, when suddenly, without the slightest warning, without any pallor of the face (which always had much colour), she fell headlong to the ground, striking her head against the floor with violence, and carrying the chair over with her in her fall. No mode of fall could have been more typical of an epileptic, as opposed to a hysteroid, attack. Instantly all the muscles of her limbs and face were jerked in most violent clonic spasm, shock-like in character, exactly such as constitutes the second stage of an ordinary epileptic fit. It involved also the spine muscles, jerking the body backwards and causing opisthotonos. She was absolutely unconscious. Her face became livid and swollen, froth came from the mouth, and she appeared as if on the point of death, just as patients often do in severe epileptic fits. After a few minutes the clonic spasm ceased, and the limbs then became rigid, and the eyeballs converged just as in a hysteroid fit. The rigidity lasted but a short time, and then she lay motionless, and it was some time before any evidence of consciousness could be obtained. The characters of this attack will be again referred to.

The attacks which most frequently consist only of clonic spasm are slight fits of partial distribution, beginning in, and often confined to, one limb. The most perfect type of these attacks are those which are sometimes produced by organic brain disease, and Hughlings Jackson has proposed to designate them 'epileptiform convulsions' as distinguished from ordinary epileptic fits. But such local attacks of clonic spasm sometimes occur in cases in which there is no other reason to believe that there is such disease as tumour, &c., and in which the variable position of the local fit, and other symptoms, preclude the supposition that the symptoms are due to any fixed, coarse, organic disease. These local clonic convulsions are especially common in the upper limb, beginning in the hand. First the fingers begin to twitch, then the elbow is jerked, and the shoulder. The attack may then cease, or the face or leg may be jerked in the same manner. Consciousness is often not lost if the spasm is confined to a single limb. Less commonly these attacks begin in the face, and still less commonly in the leg (see Chap. II.) Their march is described in p. 82.

When a severe attack begins by local clonic spasm, this, when it reaches a certain extent and degree, may pass into tonic spasm, succeeded again, at a later stage, by clonic spasm. In such cases the period at which the tonic spasm comes on, and its duration, vary; if the attack is severe, it may come on early; if slighter, there may be only a little tonic spasm in the middle of the fit.

An instance of the latter form was afforded by a girl, aged ten, who, while I was looking at her, suddenly turned her head to the right, then turned round to the right, and fell. As she fell, the right arm began to twitch at the shoulder and elbow-joint; the clonic spasm did not involve the hand, the fingers of which were extended and still; presently the face began to twitch on both sides, then both legs were similarly involved, and then the left arm. Then all parts were fixed in tonic spasm, which lasted for a few seconds, and was again succeeded by clonic spasm.

In fits which begin locally, the spasm is not always clonic in the commencement; it may be at first tonic and afterwards clonic, as in a typical attack, and this although the fits are very limited in range. In one patient, for instance, the attacks were limited to the right arm and right side of the face, and commenced by tonic flexion of the fingers, first at the metacarpo-phalangeal joints, and then at the other joints, the flexion being greatest towards the ulnar side. The thumb was inverted or pressed against the first finger. Next the elbow-joint was gradually flexed, and the arm adducted by the pectoralis, so that the hand was brought in front of the chest. After the arm had got into this position, the tonic gradually passed into clonic spasm exactly as in an ordinary epileptic fit. This patient had also some attacks which began in the face, and a few which began in the right leg. There was a probability (although not conclusive evidence) that he was the subject of cerebral tumour, but his seizures are here mentioned because they are particularly instructive as an illustration of the characters of this form of seizure. Different parts, as the face and arm, being affected in succession, often presented, at the same moment, different stages of convulsion. For instance, when the face was affected first, and the arm secondarily, there was frequently clonic spasm (second stage) in the face, and tonic spasm (first stage) in the arm. In slight attacks involving face and arm, the leg often escaped altogether, but in severe attacks it was involved in the tonic and clonic spasm. In less severe seizures the leg was the seat of tonic spas only, although the face and arm presented both forms spasm. In some of these attacks a change in the form tonic spasm in the leg was observed to occur at the ti of the change from tonic to clonic spasm in the arm. T leg was always rigidly extended, and at first the foot w inverted and the great toe pointed downwards and inward When, however, the change in the form of spasm occurre in the arm, the foot ceased to be extended and inverte the ankle-joint became flexed and the great toe ove extended so as to point upwards.

It is instructive to compare with this change in th form of tonic spasm, from extension to flexion of the ankle synchronous with the change from tonic to clonic spas in the arm, the characters of the tonic fit mentioned o p. 78. In this during the course of the fit there was corresponding change from extensor to flexor spasm.

*March of the Convulsion.*—Considerable interest attaches to the march of fits which begin locally, and of which the case just mentioned is an example. It is scarcely possible to consider this subject without referring to certain pathological questions which are involved, and which would find a more logical, although less convenient, place in the chapter on Pathology. In that place the theory here assumed, that the discharge in epilepsy originates in the cerebral hemispheres, will be further discussed.

Hughlings Jackson has drawn attention to the frequency with which the fits begin in the hand, and associated with it the law that the spasm most frequently begins in small muscles which are put to the most varied uses, and he has put forward also the hypothesis that these small muscles are represented by small cells, and that such cells are less 'stable' than larger ones. But it is important to note that (as stated in the description of modes of onset) a large number of fits beginning in the hand commence, not by a motion, but by a sensation. No doubt in these the same general principle holds good: the sensory centres in which the hand is represented are much more highly developed than those in which the upper part of the arm is represented. But the law holds good only generally, not universally, with regard to either motor or sensory auræ. The thumb and forefinger are the parts put to the most varied uses, but initial spasm, though it sometimes occurs in them, occurs more frequently in some other part of the hand, and an initial sensation is much less frequently felt in the parts of the hand in which sensation is most highly developed—the palmar aspect of the fingers—than in some other part, as the knuckles or the wrist. (See pp. 46 and 47.)

Moreover, cases are not rare in which the spasm begins in the larger muscles of the shoulder or hip. I have mentioned (p. 81) a case in which the convulsion began in the shoulder. In another patient, whose attacks were carefully watched, twitching began at the left shoulder, and the spasm descended the arm; then it began on the left side of the face, and finally affected the leg. In the leg it is very frequent for the first spasm to begin in the large muscles of the hip or thigh. But it is to be remembered that elaborateness of movement is much less developed in the foot than in the hand.

The march of the spasm, in the cases in which the commencement is deliberate and local, has been already considered at some length in the account of the modes of onset. It usually rapidly extends through one side, and then affects the other. It is partial, confined to one side, or to part of one side, less frequently in idiopathic epilepsy than in the convulsions which result from organic brain disease. Not rarely, however, it has begun to lessen on the side first affected before the second side is involved, so that it may be in a different stage, at the same time, on the two sides. When the side first affected is in clonic spasm, the side affected secondarily may be in tonic spasm. The extension to the second side is marked, as already stated, by a turning of the head and eyes away from the side first affected. The cessation of the fit is usually in the order of invasion. It ceases first in the part first affected.

The relation of the spasm to the muscles of unilateral and of bilateral use is a point to which attention has been called by Hughlings Jackson. In a unilateral convulsion, for instance, the respiratory muscles on both sides of the chest may be equally affected. A unilateral fit beginning in muscles of perfect bilateral use commonly commences in both. I have recorded[1] a case of convulsion from cerebral tumour (situated in the white substance of the hemisphere above the lateral ventricle), in which the convulsion, although unilateral, began in both frontal muscles. The orbicularis oris is a central muscle of bilateral use, and a unilateral convulsion commencing in this muscle begins on the whole.[2]

The theory of Broadbent,[3] to explain the escape of the bilateral muscles (of thorax, abdomen, jaws, eyelids, eyes, &c.) in hemiplegia, affords an equal explanation, as Hughlings Jackson has shown,[4] of their involvement in spasm. The theory is that the commissural connection between the nuclei of these bilateral muscles is so close that the two nuclei are practically fused into one, and can be called into action from either hemisphere of the brain. Underlying the theory of Broadbent is the hypothesis that the bilateral muscles are represented in each hemisphere of the brain much more equally than the unilateral muscles. We do not yet know to what extent this is subserved by a direct connection of these muscles with the hemisphere on the same side by fibres which do not decussate.

A discharge of the motor centres in one hemisphere causes a convulsion on the opposite side. If this is slight, only the muscles of unilateral use may be involved. In such convulsion the muscles of bilateral use (as the muscles of ordinary respiration) may escape, probably because the very equality of representation in the two hemispheres of the brain involves a less absolute representation in either. If the discharge is greater in degree, it involves, besides the unilateral muscles of the opposite side, the bilateral muscles of both sides, in proportion to the degree of functional association. The muscles of the thorax are affected equally. The leg on the same side may be affected, but in a slighter degree than the other. The arm usually escapes. Hughlings Jackson believes that when the spasm spreads to the whole of the second side the discharge is still limited to the one hemisphere. In some cases this may be true.[1] But the character of the convulsion, in other cases in which the spasm has involved first one side and then the other, seems to suggest strongly that there is an extension of the discharge from one hemisphere to the other. In the early stage of the fit we have deviation of the head and eyes to the side first affected, and as the spasm is lessening on this side there is a similar deviation of head and eyes to the side secondarily affected, and the convulsion on this side passes through the same stages as on the other, on which it has often ceased when it is at its height on the second side. The corpus callosum is known to connect the convolutions of the two hemispheres, and Brown-Séquard has lately demonstrated by experiment that through its fibres the motor centres of the cortex may be called into action.

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[1] *Brit. Med. Journal*, Sept. 26, 1874.

[2] Hughlings Jackson, *Med. Times and Gazette*, Jan. 6, 1872. I have also met with a similar case.

[3] *British and Foreign Med. Chir. Review*, April 1866.

[4] *Med. Times and Gaz.*, Aug. 5, 1868; and 'Localisation of Movements in the Brain,' *Lancet*, Feb. 1873.

It may be urged that in idiopathic epilepsy the affection of the two sides is synchronous, and that therefore there cannot be a passage of the discharge from one hemisphere to the other. But the instantaneous involvement of all the muscles of one side may occur from spread of discharge. In some cases minor attacks begin locally and spread slowly, and severer attacks seem to begin simultaneously in all the muscles. In the latter case we are justified in assuming that there is a spread of discharge through the centres, which occurs too rapidly to be observed. There is no greater difficulty in admitting the instantaneous spread of an intense discharge to the opposite hemisphere than in admitting its instantaneous extension through the centres of one hemisphere. So rapid is the transmission of nervous impulses, that centres structurally connected by fibres which extend from one hemisphere to the other may be in as intimate functional connection as centres which lie in juxtaposition. Luciani's experimental demonstration of what may occur in a mutilated brain does not prove that the process is always the same when the hemispheres are intact.

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[1] The experiments of Luciani (*Rivista Sperimentale de Frenatria*, iv. 1878, p. 617) in which he succeeded in producing general convulsions by stimulating the leg-centre in one hemisphere, all other cortical centres having been removed on both sides, shows that general convulsion may be produced by a unilateral discharge, possibly, as he supposes, by the subordinate agency of the medulla oblongata.

That this theory of the process of the epileptic fit is correct, is, I think, proved by a very important case which has been lately published by Oebeke,[1] and which also, as its narrator points out, is of not less value from the evidence which it affords that the convolutions are the seat of the discharge in idiopathic epilepsy. A patient who had been liable to general epileptic fits from birth was seized, in adult life, with left hemiplegia, due, as was afterwards discovered, to a hæmorrhage in the central ganglia of the right hemisphere. The epileptic fits continued to occur after the onset of the hemiplegia, but affected only the unparalysed side.

The more rapid the development of the spasm in a fit beginning unilaterally, the quicker and more equal is the involvement of the side first affected. As an exception to the rule that severe unilateral spasm commonly spreads to the other side and affects it considerably, a very unusual case deserves mention in which, in a very long continued attack, one side was involved almost exclusively, the other being the seat of slight convulsion from time to time. The duration of the attack (twenty minutes) was also very remarkable. The patient presented no evidence of organic brain disease.

The patient was a boy, ten years of age, who had been subject to fits since the age of six. There was no inherited tendency. The attacks were preceded by an aura which commenced at the lower part of the stomach with a 'winding up,' and the sensation seemed to ascend to the top of the head, when he lost consciousness. Several attacks which were seen were brief right-sided fits, in which the arm and leg became rigid, the latter being strongly flexed. He also had two longer attacks, one of which was watched by Dr. Beevor, the resident medical officer, who noted, about three minutes after the onset, that the patient was quite unconscious; the head and eyes were turned to the right, the face was jerked on the right side, the right arm was strongly flexed at the elbow, and jerked in violent flexor clonic spasm. The fist was clenched. The right leg was extended and rigid, and the seat of fine tremor. The respiratory muscles were convulsed synchronously with the arm. The pupils were both widely dilated. After this convulsion had continued on the right side for ten minutes (by the watch), and the face, at first red, had become cyanotic, the clonic spasm commenced in the left arm and leg, the latter being flexed at the hip and knee joints. After a few minutes it ceased on the left side, continuing on the right. Nineteen minutes after the onset the convulsion ceased in the right side of the face, and at twenty minutes in the arm and leg also, and then the left arm and leg were again slightly convulsed for a few minutes. In twenty-five minutes there was again slight rigidity in the left arm and leg, with deviation of the head to the left. In two or three minutes more this also ceased, and there was no return.

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[1] *Berlin. Klin. Wochenschrift,* 1880, No. 37.

*Sensation of Convulsion.*—Little is known of the sensation which attends severe convulsion, since the patient is almost always unconscious. Slight clonic or tonic spasm is often unattended by pain. When considerable it is usually very painful, and sometimes a slight amount of spasm is attended with considerable suffering. Thus one patient described the sensation before he lost consciousness 'as if his arm were being torn off.' Another, who had unilateral fits, without loss of consciousness, said that 'if he put his arm into a furnace it would not equal the pain.' Another, a girl of twenty-five, who had had fits since the age of thirteen, was often waked in the night by the attack. She would find the head turned to the left, and the left arm 'twisted,' and she said that she 'could not think that the pain of the arm being cut off would be more severe than that she then felt.' It is probable that in some cases in which the pain is very severe, and the spasm moderate, the sensation is not due to the latter, but to the discharge involving the sensory centres.

*Pupils.*—At the onset of a fit the pupils have occasionally been found contracted. Dilatation, however, quickly occurs, and is present as soon as the tonic stage is well established. It is rare for the pupils to be seen in any other condition than dilatation. This continues until the fit is over. While dilated they do not act to light. When the patient begins to exhibit slight signs of consciousness on an attempt to rouse him, the dilatation ceases.

At the end of the fit, usually a few minutes after the cessation of the convulsion, the pupils sometimes exhibit a remarkable oscillation. It was first pointed out by Reynolds,[1] and attention has been specially directed to it by Clouston and Echeverria. It is not a constant phenomenon, being more frequently absent than present. There are alternate contractions and dilatations, repeated every one or two seconds, for a few minutes.

*Reflex Action* is abolished in all severe fits, but its investigation is a matter of difficulty on account of the spasm. The absence of reflex action of the pupil has been already spoken of. The conjunctiva may be touched without any reflex contraction of the orbicularis. The loss of reflex action continues until a few minutes after the convulsion is over, but its condition after the attack will be described presently.

*Sphincters.*—During a fit there is often involuntary passage of urine and fæces, the former much more frequently than the latter. It is not merely the result of unconsciousness, since it occurs in some patients, never in others, although their attacks are equally severe. Further, urine may be passed without the patient being at the time actually unconscious, as in a case which will be mentioned in the description of minor fits. The event seems thus to be part of, or related to, the convulsive action of the fit. The same conclusion is suggested by the occasional relation of the act of micturition to the aura of a fit, as pointed out on p. 52. Passage of urine or fæces is rather more common in fits which come on during sleep than during the waking state. I have known it to be invariable in nocturnal fits, although, in the same patient, it never occurred during fits in the day. Emission of semen sometimes, but rarely, occurs, it is said, towards the end of the tonic stage.

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[1] *Epilepsy, &c.*, 1861, p. 112.

*Vascular System.*—The pulse may be feeble at the onset, but I have never noted an actual initial failure. Sometimes it is unaffected at the commencement. A tracing published by Voisin[1] shows that the heart's action may be perfectly normal during the stage of the aura. As the muscular spasm becomes considerable, the pulse is increased in frequency and often also in force. It may be 120–140 per minute. The frequency gradually lessens after the cessation of the fit, and the pulse then usually becomes feeble and small.

The colour of the face at the onset varies much in different cases. It is often pale, but the pallor is less constant than is currently asserted. It is sometimes quite unchanged, and in some cases the face is flushed at the onset. As Voisin correctly states, the pallor may be considerable after the tonic spasm is established, although there was none at the onset. After the tonic spasm has lasted a short time, ten or twenty seconds, the face becomes congested, and then cyanotic. The venous congestion may be extreme, and the bloated dusky tint of the features, distorted by the spasm, renders the aspect of the patient most alarming to those unaccustomed to the disease. With the more complete intermission of the clonic spasm, air becomes changed in the lung, and the cyanosis lessens. During the course of the convulsion the skin often becomes covered with sweat.

*Ophthalmoscopic examination* during a fit of idiopathic epilepsy has rarely been satisfactorily effected. My own observations relate only to cases of convulsions beginning locally, a class of fits in which there is rarely any initial pallor of the face. In several of these cases I have watched an artery by the direct method from before the commencement to the end of the fit (in one case during a very severe unilateral convulsion), without detecting the slightest diminution in size of the vessel. During the stage of cyanosis the veins of the retina always become distended and dark. During the status epilepticus, in which fits occur in rapid succession for several days, I once observed congestion of the discs with slight œdema, which passed away after the cessation of the series of fits. (I may here observe that during the intervals of fits the ophthalmoscope does not, according to my experience, reveal any abnormal appearances which can be associated with epilepsy.)

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[1] *Dict. Nouv. de Méd. et Chir.*, art. 'Epilepsie,' tom. xiii. p. 584.

Excitants of Attacks.—In some patients attacks may be excited by special influences. Emotion has much less effect in bringing on epileptic than hysteroid seizures, but it is sometimes distinct even in the former. Sudden noises occasionally produce attacks, usually of minor character. In one case, for instance, in which the severe attacks were certainly epileptic, and were attended with tongue-biting, minor attacks, consisting of a sudden swoon, were readily produced by a loud noise. A still more striking case was that of a young man whose attacks began by a sensory aura in the hand, passing up the arm, and in whom minor attacks, consisting of this sensation only, could be at any time produced by a sudden loud noise, such as slamming a door. A sudden 'startling' noise, as the adjective denotes, may cause, normally, a momentary discharge of the motor centres, and it is therefore not surprising that it should excite a pathological discharge when there exists a morbid instability of tissue.

In very rare instances the influence of light seems to excite a fit. I have met with two examples of this. One was a girl of seventeen, whose first attack occurred on going into bright sunshine, for the first time, after an attack of typhoid fever. The immediate warning of an attack was giddiness and rotation to the left. At any time an attack could be induced by going out suddenly into bright sunshine. If there was no sunshine, an attack did not occur. The other case was that of a man, the warning of whose fits was the appearance before the eyes of 'bright blue lights like stars—always the same.' The warning and a fit could be at any time brought on by looking at a bright light, even at a bright fire. The relation is, in this case, intelligible, since the discharge apparently commenced in the visual centre.

In some, also rare, cases attacks can be brought on by voluntary motion. A curious instance of this was presented by a boy who was lately at the Queen Square Hospital under the care of my colleague, Dr. Ramskill. The nature of the case was obscure, since the patient had, at one time, general powerlessness. As he recovered perfectly, it is to be presumed that there was no grave organic disease, in the ordinary sense of the word. At one time any passive movement of the trunk which involved a movement of the spine, at once brought on an attack of general tonic spasm, in which the legs were extended and the arms irregularly flexed just as in epilepsy, but without any clonic spasm. During the attack, which lasted only ten or fifteen seconds, there was complete unconsciousness. After he recovered voluntary power an attack occurred whenever, after remaining still for a time, he attempted to walk, but the attacks were then much slighter, and consisted only in sudden irregular fixation of the limbs without loss of consciousness. In another patient who had also severe epileptic fits, minor attacks, consisting only of giddiness and staggering, were sometimes excited by sudden movements. A similar effect was observed in a girl of thirteen who for two years had had attacks of a very unusual character, brought on by movement after sitting still. On rising from a chair, for instance, after taking four steps she suddenly made a quick movement forwards; the right arm was raised to the head, and the other down and back; the fingers of each hand were strongly extended, and the right hand made scratching movements, and did actually in some attacks scratch the face severely; her face assumed a terrified expression, the eyes wide open, the tongue protruded to the left and grasped by the teeth, but not bitten; the face was flushed at first, and gradually became pale; the pupils were widely dilated, much more so than was habitual. The attack lasted about a minute; she was confused for a moment or two; then she was quite well. But after some attacks she swung her right arm round as if turning a mangle. At a later period this coordinated movement after the attack did not occur, and the fits came on independently of movement.

Very interesting is the effect of sudden muscular tension, or the vibration of tense muscles produced by percussing their tendons, in exciting attacks. The effect is chiefly seen in cases of organic brain disease, with convulsions beginning locally. In these cases the attempt to obtain the so-called 'tendon-reflex' actions, in the limbs in which the convulsions begin, may set up an attack. Brissaud has noted, for instance, that percussion of the patellar tendon may have this effect, and Hughlings Jackson has recorded an interesting case in which attacks, commencing in the foot, were excited by an attempt to obtain the foot-clonus by sudden flexion of the ankle. I have also observed percussion of the patellar tendon to set up an attack commencing in the leg. The fact that, in some cases of organic brain disease, gentle passive movement of a paralysed limb may set up the commencement of a fit, is no doubt another example of the same phenomenon. I have recorded elsewhere[1] an interesting example of this. The right arm had gradually become weak and paralysed; there were frequent convulsive attacks, beginning in the right hand. The arm was rigid, and any attempt at passive movement, and even the muscular tension of allowing the arm to hang down, brought on the sensation of a fit. Voluntary movements of the fingers had the same effect.

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[1] *Medical Ophthalmoscopy*, p. 256.

Phenomena analogous to the 'epileptogenic zone' of Brown-Séquard's guinea-pigs—the production of attacks by irritating some part of the skin—are almost unknown in idiopathic epilepsy. The nearest approximation to it which I have seen was in a man, aged 33, who was subject to right-sided fits since a fall on the head at 22. The attacks began deliberately by spasm in the right arm, and they could be at any time produced by touching the skin at the upper edge of the *left* scapula.

Little is known of the effect of electrical stimulation in exciting attacks. In a case of cerebral tumour I was able to excite the aura, by which attacks commenced, by applying voltaism to the region of the cervical sympathetic, but whether it was by stimulation of this nerve is uncertain.

ARREST OF FITS.—Sometimes attacks which begin deliberately can be cut short, and the means by which their arrest can be effected are of considerable pathological interest and of some practical importance. Attacks which begin by a general or bilateral aura, or by the epigastric sensation, can rarely be arrested, but now and then they may be stopped by some muscular exertion, as by walking quickly about the room, or by a strong sensory impression, such as the application of ammonia to the nostrils, and sometimes by the inhalation of nitrite of amyl. The attacks which can be most frequently arrested are those which begin by a unilateral peripheral aura, especially those commencing in the hand or the foot. The most common mode by which the arrest is effected is by the application of a ligature around the limb, above the part convulsed. This method is due to Pelops, the master of Galen, who was led to employ it by his theory already mentioned (p. 40), that the aura consisted in the ascent of some vapour, analogous to a venomous poison, up the vessels, and the ligature was applied to arrest the ascent of this, just as it would arrest the ascent of the poison of a snake-bite. The ligature was frequently employed by physicians of the sixteenth and seventeenth centuries with the same object—to arrest the progress of some influence along the vessels, or, when the theory changed, along the nerves. This explanation has, however, been untenable since the demonstration by Odier, already alluded to (p. 41), that this method of arrest is equally efficacious in convulsions which are due to organic brain disease. This fact shows that the arrest must be effected, not in the limb convulsed, but in the centre in which the discharge is occurring, of which the local convulsion is the outward manifestation. The strong peripheral impression on the limb above the part convulsed probably raises the resistance in the nerve-cells of the corresponding part of the brain, and thus arrests the spread of the discharge. The effect of the ligature must first be exerted on the sensory centre, and through this on the motor centre. The two are connected intimately, and their mutual interaction must be constant, so that the condition of the motor centre is no doubt readily influenced through the sensory centre. Hence a fit may be arrested by the ligature, whether it begins by a sensation or by a motion, *i.e.* whether the sensory or motor centre leads in the discharge. Additional observation is necessary to determine whether the ligature is more effective in one class of fits than in the other. If the commencing fit has got beyond the part to which the ligature is applied, the attack is not arrested. The reason why a ligature is especially effective is probably partly because it is readily adopted, and partly because the cutaneous stimulation is applied to the entire circumference of the limb, and so influences the whole extent of grey matter in which the discharge is advancing. Occasionally, however, a more limited cutaneous stimulation, as a pinch or a prick, has the same effect.

It is instructive to note the antagonism which seems to exist between those afferent impulses that are excited from the surface in cutaneous stimulation, and the afferent impressions which come from the muscles when they are extended, as in the attempt to obtain the contractions commonly called 'tendon-reflexes,' the knee-jerk and foot-clonus. It has been already pointed out that one effect of this deep stimulation may be to excite a fit. On the other hand, the effect of the stimulation of the skin may be to arrest the fit. Some years ago a man was under my care whose fits began in the foot. The sensation with which the fits commenced could be at any time produced by percussing the patellar tendon, and then could be arrested by pinching the skin of the leg. A similar antagonism may be sometimes seen in disease of the spinal cord, attended by a spasmodic state of the limbs—spastic paraplegia. The paroxysms of spasm, broken by tremor, which often occur in that disease, may usually be at once arrested by a strong cutaneous impression. Thus in both cord and brain the 'discharge' may be arrested by the same means.

The repeated arrest of fits by the ligature may produce a permanent effect. I have a patient whose fits always commence in the hand by a sensation which passes up the arm, and could be arrested by the ligature applied just above the elbow. After employing the ligature in this manner for some months, he found that the fits, commencing in the same way, stopped of their own accord at the spot at which they had been repeatedly arrested by the ligature. They never stopped thus before the patient commenced the use of the ligature, which appeared to have produced a permanent increase of resistance at a certain part of the unstable nerve tissue.

If this explanation of the mode of action of the ligature be correct, it is evidently important that every fit should, if possible, be arrested, since a single passage of the barrier by the discharge might, so to speak, sweep away any functional resistance which had been established there, and render the subsequent arrest of fits more difficult. An illustration of this is afforded by the case of cerebral tumour recorded by Odier and before referred to.[1] The convulsive attacks were at first local, beginning in the little finger of one hand, and ascending at first only to the wrist, then to the elbow, and ultimately to the shoulder. The patient learned to arrest them by a ligature round the wrist, and did so for a long time. One day he omitted to apply the ligature, and the attack went on into a severe general convulsion. The commencing fits could never afterwards be arrested by the ligature.

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[1] *Man. de Méd. Pratique*, 1811, p. 130.

The fits which begin by spasm may often be effectually stopped in another way—by forcibly preventing the movement, as was pointed out by Aretæus,[1] and especially insisted on by Maisonneuve in the beginning of this century. For instance, in one patient the attacks commenced by forcible closure of the right hand. If he could succeed in forcing the closed hand open by the other hand, the fit always ceased; if he failed, the spasm went on up the arm, and he lost consciousness. In another patient the fits began with cramp in the thigh, which passed down to the heel, the leg quivering. As soon as it reached the heel, the leg was bent forcibly on the thigh, and the thigh on the body. If the patient could succeed in preventing this forcible flexion of the leg, in keeping it down, the fit ceased. If he failed to do this, the attack went on to a severe convulsion.

To this influence also an analogue may be found in the action of the spinal cord. The muscular cramp, such as occurs in the calf in the frequent experience of most persons, is no doubt due immediately to the over-action of certain nerve-cells of the spinal cord. If the contraction of the muscle can be prevented, as by pressing the ball of the foot against some object when the cramp is commencing, it passes off. Here we have (as Herpin[2] pointed out) the same phenomenon. The extension of muscles arrests, no doubt by its influence on the centre, the discharge of the motor nerve-cells of the spinal cord, just as it does the discharge of the motor nerve-cells of the brain in a commencing epileptiform seizure.

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[1] 'Whenever the disease occurs, and has already seized the finger, or is commencing in any part, [the patients] having from experience a foreknowledge of what is to happen, call, from among those who are present, upon their customary assistants and entreat them to pull aside and stretch the affected member.'—Aretæus, Syd. Soc. Trans., p. 244.

[2] *De l'Epilepsie*, 1852, p. 603.

But we have seen that, if there is no fit in progress, muscular extension, and the vibration of tense muscles, may set up an attack. Here we have an antagonism between the action of the same stimulus, probably in different degrees of intensity, acting in different states of the nerve-centre. This form of antagonism is familiar to physiologists. A slight peripheral impression, for instance, will set up a reflex action, while a stronger impression of the same kind will arrest or prevent all reflex action. Passive muscular tension excites tonic contraction in a muscle (as Tchirjew has shown), and this action may in abnormal conditions be excessive, as in the 'myotatic' contractions (so-called tendon-reflexes [1]). It has an effect on an unstable cerebral centre similar to its effect on the spinal centre, and excites the epileptiform discharge. But if this centre is in process of discharge, as in cramp, a similar extension of the contracting muscle may arrest the discharge in the related centre. The afferent nerves, it is to be noted, commence in the fibrous tissues of the muscle, and seem to be stimulated especially by extension.

In some cases, rubbing the limb in which the attack is commencing will arrest the fit, a fact which patients often discover for themselves. Almost all forms of spasm, even the 'late rigidity' of hemiplegia, and the rigidity of 'spastic spinal paralysis,' may be lessened by rubbing, and we have another illustration of the fact that influences which lessen the discharge of force from lower centres have a similar effect on higher centres.

CONDITIONS AFTER ATTACKS.—*Sleep and Headache.*—The coma into which the patient usually passes at the close of an epileptic fit often continues as heavy sleep, lasting for a quarter of an hour or longer, sometimes for hours. After the first few minutes, however, the patient can be roused. The sleep is usually followed by a severe headache, general, lasting for several hours more, and often continuing during the rest of the day. Both sleep and headache may be absent. Occasionally the sleep is not succeeded by headache, but if the patient is roused, and is not allowed to sleep, the pain in the head is severe.

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[1] See note on p. 100.

*Paralysis.*—A fit of epileptic type may leave paralysis, very rarely general, commonly of one half of the body, or of still smaller range, affecting a limb only. The form and nature of the paralysis differ essentially in different cases, and by many writers the varieties have not been distinguished. The first and most common is transient weakness, incomplete in degree, passing away in the course of a few hours, but usually recurring after other attacks, and, therefore, probably of 'functional' nature, and a direct result of the attack. The second form is that in which paralysis succeeds the first fit, or first series of fits, and persists for weeks or months or permanently, irrespective of the subsequent convulsions. These are cases in which the paralysis is the result of an organic lesion of the brain, of which the convulsions are also the consequence. They are, for the most part, cases of 'post-hemiplegic convulsion.' The third class are those in which persistent paralysis follows a fit in a confirmed epileptic. In these cases, which are extremely rare, a cerebral hæmorrhage has probably resulted from the vascular strain during the fit.

The rarity of cases of the third class renders them of small practical importance. I have never met with an example of this form. The second class, 'post-hemiplegic convulsions,' will be considered separately. The first form, the transient paralysis, which is apt to recur, alone needs consideration among the conditions after attacks.

The motor weakness, which immediately follows a fit, is sometimes distinct when a fit is from the first bilateral. It is then general. The patient may, for some minutes, be unable to walk or even to stand. It rarely, however, lasts long, and is commonly regarded as general exhaustion or prostration from the severity of the convulsion, although it may probably, as Hughlings Jackson has suggested,[1] be due to the same cause as some of the weakness which succeeds more limited seizures. After convulsions which are partial or unilateral, or which begin unilaterally, motor weakness is both more conspicuous and actually greater than after general convulsions. It usually affects most the limb in which the convulsion begins, and, as the commencement is much more frequent in the arm than in the leg, the weakness is usually greatest in the arm. It is the 'epileptic hemiplegia' of Todd, who gave an excellent description of it in one of his lectures.[2]

The duration of the weakness is not considerable, a few hours, or rarely a day or two. The degree varies; it is in most cases incomplete, slight power of movement remains. This is almost always the case in the leg. In the arm, however, the paralysis may be at first absolute, but power returns in the course of a few hours. The same weakness may be left by every fit. If the convulsions recur at short intervals, say every few hours, the paralysis may persist until the series of attacks is over. Sometimes a relation can be traced between the degree of convulsion and the degree of subsequent paralysis, but this is not always the case. In the class of fits which are most frequently succeeded by weakness, those which commence in the hand or the foot, paralysis often succeeds an attack which is extremely slight, and even one in which convulsion may be entirely absent.

It will be convenient to consider here the probable nature of the paralysis which succeeds epileptoid seizures, but before doing so certain other conditions found in the limbs after fits may be described. A severe attack leaves the muscles flabby and apparently atonic, and during this condition, in some cases, when the fit has been very severe, the knee-jerk ('patellar tendon-reflex') cannot be obtained for a few seconds. The 'myotatic irritability' of the muscles is lost.[1] This loss was first pointed out by Westphal, and I can confirm it by previous independent observation. At the end of about half a minute the jerk can again be obtained. More frequently these myotatic contractions are found to be excessive after a fit, so that the foot-clonus can be obtained, although usually only during the first few minutes. This was first pointed out by Hughlings Jackson[2] in a case of unilateral convulsion, probably due to organic brain disease. Some observations which I have made on this point, and a larger series kindly made for me by Dr. Beevor, Resident Medical Officer at the National Hospital for the Paralysed and Epileptic, have shown that the foot-clonus is usually to be obtained for a few minutes after all severe epileptic and epileptiform fits. There may thus be three conditions of these phenomena. After slight attacks there may be no change in the myotatic contractions; after fits of greater severity there may be foot-clonus and increased knee-jerk, and after very severe fits there may be, for a very short time, a loss of the knee-jerk, which is followed by a stage of excess. The relation of the excess to severity of fit is clearly shown by the fact (ascertained by Dr. Beevor) that after a general convulsion, in which each side is apparently affected with equal severity, but in which the deviation of the head shows that the convulsion is really more severe on one side, the clonus can often be obtained on the side to which the head deviates, and not on the other.

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[1] *Brain*, April 1881, p. 439.

[2] *Clinical Lectures*, edited by Beale, 1861, p. 730.

Reflex action in the limbs (*e.g.* from the sole of the foot) is abolished for five or ten minutes after a severe fit.

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[1] By the term 'myotatic' is meant the irritability which is developed by tension (*τατικός*, extended), and which is demonstrated by the contractions which occur when tendons are percussed, or in other ways mechanical stimuli are applied to the tense muscles, and cause local contractions, *e.g.* the knee-jerk or foot-clonus. The phenomena are commonly called 'tendon-reflexes.' I have described at length the evidence regarding their nature in *Diagnosis of Diseases of the Spinal Cord*, 2nd ed.

[2] *Med. Times and Gazette*, Feb. 12, 1881.

Another occasional post-epileptic paralysis is partial loss of speech. It occurs especially after right-sided seizures, and is often associated with right-sided weakness, just as is permanent aphasia with right hemiplegia. But it may occur without weakness, especially after slight fits commencing in the face (see p. 46). This defect varies much in degree, and, as Hughlings Jackson[1] has pointed out, there is more unintelligible mumbling than in cases of permanent aphasia. It is a mixture of defect in articulation as well as a defect in the cerebral speech-process.

In discussing in this place, for the sake of convenience, the probable pathology of the weakness after convulsions, it must be assumed, in anticipation of the chapter on 'Pathology,' that the discharge which constitutes the fit is that of cerebral motor centres.

Todd taught that the 'epileptic hemiplegia' was due to exhaustion of part of the brain by the excessive action, and the same theory has been accepted by Hughlings Jackson and by A. Robertson of Glasgow. The former has suggested[2] that the weakness and foot-clonus may be the result of the exhaustion of the fibres of the internal capsule of the brain or lateral columns of the cord by the 'discharge' through them, since in organic disease the occurrence of this symptom is especially related to sclerosis of these columns. The hypothesis affords a very probable explanation of some of the weakness after very severe fits, and of the excess of myotatic irritability in these cases; and the loss of the knee-jerk, which is found immediately after some fits, may be due to the initial exhaustion of the lumbar centres of the cord. But it is not easy to explain on this theory all the phenomena of post-epileptic paralysis. It is difficult to explain as an effect of 'exhaustion' the extreme weakness which may exist in the arm, and occasionally in the leg, after a slight local fit. The difficulty is increased if we regard the exhaustion as affecting chiefly the nerve fibres, which are not readily exhaustible to the extent of causing such loss of power as is sometimes observed. There is also a disproportion between the severity of the fit and the subsequent paralysis. A patient may have a severe general convulsion, and, a few minutes later, may have no conspicuous weakness, while another patient, who has had a fit confined to one arm, apparently slight in degree, may be unable to move the limb for an hour or more. Dr. Jackson has suggested, as an explanation of this discrepancy, that the sudden violent discharge is diffused widely, overcoming widely the resistances, by its force; while the slower local discharge, having more 'momentum,' does not diffuse itself, is concentrated on certain parts, which are thus stimulated for a longer time and in greater absolute degree, and so become exhausted. But there is no evidence that a slight discharge of long duration exhausts the nerve elements more than a severe but brief discharge. Moreover, a comparison of a series of cases shows that there is no relation between either the duration or degree of convulsion, or both, and the subsequent weakness; that on the one hand we may have severe or long-continued motor spasm with very little subsequent weakness; while, on the other hand, great loss of power may succeed other attacks in which there is *no* motor convulsion, in which the fit consists of a purely sensory discharge. Since the point is of considerable importance, it may be well to mention the facts of some cases in illustration.

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[1] Art. 'Convulsions,' Reynolds's *System of Med.* vol. ii. 2nd ed. p. 278.

[2] *Brain*, April 1881.

A lad, aged 20, had suffered for ten years from seizures of the following character. Each commenced with a pain in the front of the right shoulder, which passed down the arm 'like a knife cutting it,' but without any spasm. Then twitching occurred at the angle of the mouth on the right side, the tongue seemed to swell, and he mumbled and did not speak intelligibly. This lasted for a few minutes, and then the attack was over. When the spasm in the face commenced, the pain in the arm always ceased. As soon as the pain commenced in the shoulder, the arm became so weak that he could scarcely move it, and the weakness continued for about a quarter of an hour.

Another patient, a young man aged 25, had had right-sided convulsive fits since measles at six years of age. He presented no sign or history of hemiplegia. Besides the attacks of convulsion he also suffered from minor seizures characterised by sudden 'mumbling,' the use of wrong words, and weakness of the right arm, without any convulsion.

Again, a woman, aged 40, probably the subject of organic disease, having slight right-sided weakness and hemianæsthesia, has also attacks of the following character. They begin with 'ticking in the right ear,' and this is followed by a painful sensation 'like hot needles running into the skin,' which passes down the side to the leg and foot, and, after reaching the toes, it returns up the leg and side, and is felt in the arm, hand, and tongue. She mumbles and cannot speak for half an hour. There is no motor spasm, but as soon as the sensation is felt in the leg and arm, the side becomes extremely weak, so that, although ordinarily able to walk fairly well, she becomes unable to stand and scarcely able to raise the arm.

Several other cases in which sudden loss of power constituted the minor seizures will be mentioned in the chapter in which these attacks are described.

In these cases, then, we have transient paralysis without any motor spasm, *i.e.* we have lowered activity of the motor centres, but without any discharge in them. We must regard the centres as restrained or inhibited, and it is readily conceivable that a discharge in the related sensory centre should, under certain circumstances, cause such inhibition, just as a painful cutaneous impression will often inhibit or arrest reflex action, a fact well known to physiologists. If such paralysis accompanying a sensory aura is to be thus, and thus only, explained, it becomes most probable that the weakness which succeeds a slight attack of local spasm is, in part at least, of the same character.

Another interesting fact, to which Hughlings Jackson has called attention, also affords strong evidence that inhibition, rather than exhaustion, is the chief element in the paralysis after local seizures. It is that the paralysis may be greater if a commencing fit is arrested by the ligature, than if it runs its usual course. The ligature must be regarded as acting on the sensory centre and increasing the resistance in, *i.e.* inhibiting, the related motor centre. That the subsequent paralysis should be thus increased affords strong confirmation of the view that it depends chiefly upon inhibition, while it is incompatible with the theory that it depends solely on exhaustion. The latter, after very severe fits, may, and doubtless does, cause some weakness, probably, however, of very transient duration.

The phenomena of some attacks show that, although the conditions of discharge and inhibition are antagonistic and, in high degrees, incompatible and mutually exclusive, yet in partial degrees they may coexist. A centre may be in a state of partial inhibition and partial discharge, and either condition may preponderate and ultimately obtain exclusively. It is no doubt difficult to understand the possible mechanism of such conditions, but we can explain in no other way such cases as that of one patient who, in his attacks, after very slight and transient spasm in the hand, felt as if the arm were being raised above the head in violent spasm, while it was really hanging powerless by his side. Still more striking phenomena of the same kind are those presented by some cases of visual aura, described on p. 65, where this explanation of combined inhibition and discharge has been already suggested. Many of the inhibitory phenomena of the nervous system, *e.g.* of reflex centres, show that antagonistic abnormal states may coexist in incomplete degree.

Whatever be the exact nature of the temporary paralysis after convulsion, it is noteworthy that the transient condition of the limbs is precisely similar to the permanent condition in cerebral paralysis. There is motor weakness, lowered cutaneous reflex action, excess of the myotatic contractions (tendon-reflexes). When unilateral convulsions occur with great frequency, so that recovery from the weakness caused by one does not occur before another convulsion comes on, a condition of persistent absolute hemiplegia may result, having all the characters of that due to a destructive brain lesion. In a case lately under my care a patient had on three occasions, for about ten days, more than a hundred unilateral fits daily, and complete hemiplegia with inability to speak. On the cessation of each series of fits the hemiplegia passed completely away.

*Automatic and Hysteroid Phenomena.*—After epileptic fits of moderate severity the patient may pass into a condition of mental automatism, in which various acts are performed in an apparently conscious manner, but of which no recollection is afterwards retained. In other cases a patient, after recovery from the epileptic fit, passes into a state of hysteroid convulsion. Both these post-epileptic conditions may occur after major attacks, but rarely when these are very severe, and they are more common after slight attacks. In the description of these, the automatic actions will be more fully considered, while the consideration of the post-epileptic hysteroid phenomena will be best postponed until the attacks of this character are specially described.

*Temperature.*—A single fit, although severe, may cause no elevation of the temperature. Sometimes the temperature is raised half a degree or a degree above the inter-paroxysmal temperature. But when a series of severe general fits occurs, separated by short intervals, the temperature may be raised several degrees. This is seen in the 'status epilepticus,' in which severe convulsions rapidly succeed one another and are attended with considerable elevation of temperature. According to Bourneville, who has carefully studied these cases, in the severer forms of the status epilepticus, which usually end in death, the temperature may rise to 105° or 107°.

*Extravasations.*—The severity of the strain on the vascular system during a severe fit may lead to the rupture of small vessels and effusion of blood. It occurs most readily in the conjunctiva, where effusions of blood from this cause are not infrequent. It is remarkable that the vessels within the eye seem never to give way. In cases in which there has been a subconjunctival extravasation, I have repeatedly looked for retinal hæmorrhages, but always without success. Occasionally minute punctiform extravasations occur on the face. I have never seen them in any other part of the body. The occurrence of the extravasations on the face and not elsewhere may perhaps be due to the constriction of the turgid neck, by the clothes, multiplying the effect of the mechanical congestion. Little is known of the occurrence of internal hæmorrhages during severe epileptic fits. After death from convulsions they are not infrequent in the pericardium, the surface of the brain, and outside the membranes of the spinal cord; but symptoms indicating their occurrence in convulsions which have not proved fatal are extremely rare.

*Vomiting* occasionally occurs after fits, but is not common. It is, however, a dangerous symptom, since the food is apt to get into the glottis. For instance, a man aged fifty-one, who had had many severe fits for three years, had an attack one day, twenty minutes after dinner; when the fit was over, he vomited and died almost immediately, probably from asphyxia thus caused. Another symptom referable to the stomach, which is sometimes met with after fits, is extreme hunger. It is especially common in lads. The patient eats voraciously when he has recovered from an attack, although he may have had a full meal an hour before.

*Urine.*—The urinary secretion is rarely altered. Occasionally a trace of albumen is to be found in the urine first passed, and, it is said, in extremely rare cases, a trace of sugar. The frequency with which albumen is present has certainly been greatly exaggerated. Huppert[1] has stated that its occurrence is almost invariable, and that hyaline casts can frequently be found, but most subsequent investigators have failed to corroborate his assertion. Of a large number of cases in which it has been examined at the National Hospital for the Paralysed and Epileptic by myself or the several resident medical officers, it has been very rarely that the slightest trace of albumen could be detected by the most careful examination, and in no instance has there been any sugar that could be detected by Fehling's test. I found, however, in a case in which there was organic kidney disease, that the amount of albumen was distinctly increased after the fits. Dr. Beevor lately examined for me the urine after forty-two attacks in twenty-three patients. In only one instance did he find a trace of albumen, and in this case, after another attack, none could be found.

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[1] *Archiv für Psychiatrie*, 1877, p. 189.

The amount of urea excreted was found to be increased after a fit by Echeverria,[1] but Gibson[2] found 'no constant change in the urine.' That the quantity of urea is not necessarily altered is clearly shown by the following estimate which I made of the daily excretion in a case in which very frequent and severe fits occurred. The observations were made on consecutive days.

| Number of fits per day | Urea in grammes |
|---|---|
| None | 20 |
| One | 23·6 |
| Two | 18 |
| Several | 13 |
| Three | 14 |
| Ten | 14·5 |
| Five | 13·75 |
| Two | 13·8 |

*Body-weight.*—It has been stated by Kowalewski[3] that every epileptic fit causes a loss of weight, which may vary from one to twelve pounds, and is greater the more recent the disease and the more severe the attack. In the *status epilepticus* it may amount to fifteen pounds, even when the individual attacks are not severe. The loss of weight is said to be rapidly regained. On this point I have no personal observations to offer.

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[1] *Loc. cit.* p. 288.

[2] *Med. Chir. Trans.* 1867, p. 75.

[3] *Archiv für Psychiatrie*, 1881, Bd. xi. Heft 2.

# CHAPTER IV.

## *SYMPTOMS CONTINUED.*

### MINOR ATTACKS.

THE minor attacks of epilepsy vary much in character, and hence patients speak of them under various designations, such as 'sensations,' 'faints,' 'losses,' 'turns,' 'giddiness,' and, in America, 'spells.' Hence also their epileptic nature, and their connection with severer attacks, are often not known to the patients or their friends. It is not always easy to ascertain their exact character on account of their very short duration. Their onset may or may not be attended by any sensation of which the patient is conscious, and when it is not so attended, the difficulty of ascertaining their character is increased. In the majority of cases the patient is unconscious during the attack, but in a few instances consciousness is retained, although it is probably always modified in some degree.

The most common form is transient loss of consciousness without conspicuous convulsion. A patient suddenly stops for a moment in whatever he or she is doing, very often turns pale, may drop whatever is in the hand, and then is better. There may be no visible spasm, or there may be a slight stoop forward, or a slight quivering of the eyelids. The patient may or may not fall. Pallor of face is by no means invariable; I have watched many such attacks in which there was not, from first to last, the least change of colour in the face. In some of the cases in which pallor does occur, the change of colour is not always general. The cheeks may not change colour at all, although the lips and the skin around become strikingly pale. There is rarely any cyanotic tint. Sometimes, as an attack is passing away, the face becomes flushed, and this whether there has or has not been any initial pallor. The attack usually lasts only a few seconds. The return of consciousness may be sudden, and the patient, after the momentary lapse, may be in just the same state as before the attack, may even continue a sentence or action which was commenced before it came on, and suspended during its occurrence. Very often, however, the patient is left in a more or less confused state, which slowly passes away. When full consciousness is suddenly regained, although it had been for a second or two completely lost, the patient may not know of the occurrence of the attack, and only become aware of it by finding that persons are looking at him with surprise. In many cases, however, some warning sensation precedes the loss of consciousness, and of this only the patient is conscious, and it is for this reason that the attacks are often termed 'sensations.' This warning may be the same as that which, in the same case, precedes the major attacks, but in many patients there is no such correspondence. Usually the sensation of which the patient can give an account is merely the aura preceding an actual loss of consciousness. To the patient the former, to the friends the latter, constitutes the evidence of the attack. In rare instances, in which consciousness is not lost, the sensation experienced by the patient constitutes the only symptom of the attack.

The following list comprehends the information which could be learned regarding the characters of the attacks in 155 cases. In many of these the characters of the attacks could only be ascertained from the patient; and hence the list conveys information as to the subjective rather than the objective symptoms.

| | |
|---|---|
| Sudden momentary unconsciousness, or 'fainting,' or 'sleepiness,' without warning | 45 |
| Giddiness | 25 |
| Jerks or starts of limbs, trunk, or head | 17 |
| Visual (sensation 10, loss 7) | 17 |
| Mental state (sudden sense of horror, &c.) | 8 |
| Unilateral peripheral sensation or twitching | 7 |
| Epigastric sensation | 5 |
| Sudden tremor | 4 |
| Sensations in both hands | 3 |
| Pain or other sensation in the head | 3 |
| Choking sensation in the throat | 3 |
| Sudden screams | 3 |
| Olfactory sensation | 2 |
| Cardiac sensation | 2 |
| Nasal sensation | 1 |
| Sensation in eyeball | 1 |
| Sudden dyspnœa | 1 |
| Sudden dropping of jaw | 1 |
| Momentary general rigidity | 1 |
| Lateral deviation of head | 1 |
| Sudden retching | 1 |
| ther general 'indescribable' sensations | 3 |
| | 155 |

Of the warning sensations, giddiness is thus the most common—a sudden sense of turning, or of the movement of neighbouring objects, or merely a vague sense of defective equilibrium, not amounting to actual 'vertigo.' Although giddiness is one of the most frequent warnings of these attacks, it is present in only a small proportion of the total number, in not more than twenty per cent. Hence it is an error to employ, as is sometimes done, the term 'epileptic vertigo' as a general designation for attacks of minor epilepsy. With or without vertigo there may be actual rotation. One patient would suddenly, without any change of colour in the face, turn half round to the right, and seem as if falling to the right, and then recover herself, and begin muttering and pulling objects off the table for a few moments, and then was better. In another case, any object to which the patient was looking seemed to move to the right, and she tried to follow it, looking more and more to the right until she had turned quite round.

Visual disturbances are also very common, either an actual visual sensation, a flash of light or stars, a sensation of colour, or else sudden loss of sight. The former is twice as common as the latter. Auditory sensations are less common, olfactory warnings still less frequent.

The epigastric sensation, which is so frequent an aura of the major attack, is comparatively infrequent in the minor seizures. Other occasional sensations are constriction of the throat and dyspnœa, and cardiac sensations. A patient who had distinct epileptic fits, in which urine was passed, had minor seizures, which consisted only in sudden palpitation of the heart and a sense of choking. A sensation in both hands or both feet, a feeling of malaise, or some other general sensation, is not uncommon; very often the patient can give no other description than that 'I feel queer all over.' Some patients scream at the onset of the seizures in a quasi-volitional manner, but without afterwards remembering it, and now and then the scream is repeated two or three times. But the patient also may scream before consciousness is lost. One patient could always hear herself scream, but could not prevent the cry.

Urine may be passed in attacks in which there is loss of consciousness, but micturition is far less common than in the major fits. As already mentioned, in rare cases urine may be passed when the patient is not completely unconscious, as in one patient whose minor attacks consisted in sudden giddiness, objects going to the left, and a feeling of fear. She always passed urine in the attack, and knew when she did so, but could not prevent it.

Sudden starts or jerks constitute a very common form of *petit mal*, sometimes with loss of consciousness, sometimes without. In the latter case it may be doubted whether they deserve to be ranked as distinct seizures. For instance, one patient who had occasional epileptic fits was liable to these starts every morning, soon after getting up. Both arms jerked suddenly, and apparently the legs also, since, if she was standing at the time, she would fall.

Occasionally the start will throw the patient forward on the knees. The start may be followed by transient dulness, though there is no actual loss of consciousness, as in one patient, whose major attacks were severe, and who had minor attacks, one of which I witnessed. She gave a sudden start, put her hand to her head as if in pain, seemed to have a difficulty in speaking for a moment, and then was better. There did not appear to be a moment of actual unconsciousness. Closely allied to the starts is a sudden backward movement. One patient, a girl, had had for fifteen years many attacks every day in which she suddenly took a step or two backward, looking strange for a moment, and immediately was well. She never fell unless there was some object behind her to trip her up. There was complete unconsciousness for the moment. She was never aware of the attack, except by finding that persons near were looking at her in astonishment. I witnessed many of these seizures, and neither I, nor the students who were with me, could ever discern the slightest change of colour in the face, which was fairly ruddy. Another patient, in the minor attacks, always suddenly went back, the face was drawn slightly to the right, consciousness was lost for a moment, and then he was better.

A similar slight drawing of the face on one side is not uncommonly the only indication of spasm. But in the case of fits which begin locally in one limb, the minor attacks usually consist solely in the spasm, or sensation, with which the major attacks commence, often without any loss of consciousness. Sometimes there is local spasm of this kind in cases in which the severer fits are so rapid in their development that the convulsion is apparently at once universal, and loss of consciousness is so early that there is no warning. One patient, for instance, who had such severe epileptic fits, had also slight attacks in which, as in one that I witnessed, she suddenly began looking around and mumbling, without the least change of colour, then lay back in the chair in which she was sitting; the right arm was stretched out, then became stiff, with the fingers flexed; respiration was difficult for a moment, and the pupils became larger. Then the attack was over; but she seemed stupid, and could not be made to speak for a few minutes.

In some of these cases the spasm may be variable in its seat. A man had at first very slight attacks, consisting of sudden rigidity of some part of his body, rendering it immoveable, sometimes one limb, sometimes the jaw, and each lasting, he conjectured, five or ten minutes. He had also other minor attacks characterised by 'dizziness.' He subsequently became subject to severe general convulsions.

Another form of minor attack, in patients whose severer fits begin unilaterally, consists in sudden weakness of the limb in which the severer attacks commence. For example, a patient who was liable to severe left-sided fits preceded by a throat aura, had also minor attacks which consisted in sudden giddiness and inability to move the left side for a few minutes. Another patient, a man aged 28, without history of syphilis, had suffered for three years from fits which commenced in the right side of the face (eye and angle of mouth); then consciousness was lost, and the right arm and leg were convulsed, and afterwards the left limbs. For a day or two after each fit his speech was altered, and the right arm and leg were weak. He had also minor attacks which commenced by a noise in the throat, and consisted in inability to speak and inability to move the right arm and leg, there being no convulsion. These cases have been already alluded to (p. 102) on account of the evidence they afford that weakness associated with epileptoid seizures may in minor attacks be due to inhibition of the centres which are discharged in severer attacks.

In some cases, not very common, the minor attacks are characterised by a sudden sensation of fear or horror. One patient, for instance, an adult man, always screamed 'Oh, it is dreadful!' at the top of his voice, and then covered his face with his hands as if horrified; in about three seconds he was all right again, and remembered nothing of the attack.

In many of the cases in which the slight attacks are attended by a definite and uniform sensation, the severe fits occur without warning. This is especially the case when the minor fits consist of vertigo only. When the severe attacks are attended by a definite aura, this, as already stated, may or may not correspond to the sensation which attends the minor attacks. For instance, one patient whose severe attacks began with a sensation in the eyeballs as if they were being pushed back into the head, and rotation of the head to the right, had minor attacks consisting in a sudden sensation of sparks before the eyes. Another patient, whose minor attacks consisted in a 'sudden smell like sulphur which seemed to stir up from the pit of the stomach' had also severe attacks of which the warning was a sudden sound. Another patient, whose major attacks were preceded by a sensation of 'crawling' on the soles of the feet, which passed up to her head, had also minor attacks which consisted in a sudden sense of extreme fear.

It is, however, much more common, when the major attacks are preceded by a definite warning, for the slight fits to be preceded by, or to consist of, the same sensation. This may, however, be combined with some other warning. For instance, a patient whose severe attacks were preceded by a sense of alarm had slighter attacks consisting of the same sensation of alarm combined with giddiness.

The sensations which attend the minor attacks are not always uniform, but may differ in the same patient at different times. A girl, whose severe fits commenced by a burning sensation at the epigastrium and sense of choking, and were distinctly epileptic, attended by tongue-biting, had slighter seizures, which consisted at first in a burning pain at the epigastrium and lower part of the sternum, lasting a few minutes and followed by sickness, but at a subsequent period they consisted in transient loss of sight, followed, as sight was returning, by dazzling sparks of colour, especially red.

CONDITIONS AFTER MINOR ATTACKS.—In many cases consciousness is obscured for a few minutes after a minor fit; the patient remains 'dazed' and stupid, answering questions with difficulty, and sometimes going to sleep. In other cases the patient, during this stage, performs some actions automatically, or passes into a condition of hysteroid convulsion. Both these post-epileptic phenomena may, as already stated, occur after severe fits, but are more common after attacks of *petit mal.* They are of great importance, and must be considered in detail.

*Automatism.*—The automatic action is often regarded as constituting the epileptic seizure, and the attacks presenting this feature have, following Esquirol, been called 'masked epilepsy,' or 'epilepsia larvata' after Morel. Whether this mental automatism may replace and represent an epileptic seizure, or whether it is always a post-epileptic phenomenon, is a question still undecided, which will be presently considered.

It is certain, however, that the automatic state often succeeds an attack, and is then essentially a post-epileptic phenomenon. According to an hypothesis suggested by Anstie and Thompson-Dickson, and applied by Hughlings Jackson, it is the result of the exhaustion of the highest cerebral centres by the discharge, and the consequent temporary loss of the control which these should exercise over complex centres just below them, which consequently act in an insubordinate and automatic manner. This theory will be again alluded to in the chapter on 'Pathology.'

These automatic actions are not merely of clinical interest, but also of practical importance on account of their medico-legal aspect, since they are sometimes complex and have the aspect of voluntary actions. It is, indeed, often not easy to convince observers that these actions are not deliberately volitional and intentional, so apparently conscious are the patients; but consciousness is in an abnormal state, for the memory retains no recollection of these actions.

In the simplest form, a patient, after an attack, seems dull and stupid for a moment, and then proceeds to perform some inapposite action in a dreamy manner. A very common action is that of undressing, and it occasionally has serious practical inconveniences. One of my patients, for instance, was in the habit of giving lessons in music, and had to relinquish his occupation, because, while giving a lesson to a young lady, after an attack so slight as to be unnoticed by his pupil, he suddenly began to take off his clothes. The act of undressing is perhaps the result of a sensation of illness which suggests going to bed. Another occasional action which may be the result of the same suggestion, is an attempt to walk up a flight of stairs which the patient thinks is before him. Thus one man, who had his fit in a kitchen, thought that the shelves of a dresser were stairs, and tried to walk up them. Another patient had a slight fit at dinner, and immediately stepped on to the table and made movements as if going upstairs. Usually, as in this instance, the action is a simple and natural one, only rendered equivocal by the surrounding circumstances. Another similar action is to put into the pocket any object that may be near, irrespective of its ownership. A young man who presented this symptom was a draper's assistant. He had minor attacks which consisted only in brief loss of consciousness, preceded by an olfactory aura—a 'nasty, indescribable smell in his nose and mouth, a sort of combination of smell and taste.' After the attacks he constantly found in his pocket any object which had been near him when the attack came on, such as scissors, reels of cotton, &c. This patient had always a sensation of hunger after the attacks, and he occasionally only discovered that he had had a seizure by finding, to his surprise, that he was cutting bread and butter and eating it as fast as he could. It is a very common thing for patients in the hospital, after slight attacks, during the automatic state, to go to other patients' lockers, take things out, and put them in their own pockets. One woman, whose general conduct suggested no suspicion of dishonesty, after an attack went to the locker of another patient, took out a purse, and pocketed it.

Occasionally the actions performed are extremely complex. I have known, for instance, a carman, after an attack, to drive through the most crowded parts of London for an hour without an accident, and retain no recollection of it afterwards. I have even heard (from Dr. Radcliffe) of a young lady playing, during this state, the most difficult music without mistake. Trousseau relates the case of an architect who, when seized on a scaffold, would run quickly from plank to plank for a few seconds shouting out his own name. He never fell. The state is no doubt closely allied to the condition in somnambulism, in which the precision of muscular action is well known.

Occasionally the automatic actions display emotion, and, it may be, violence and rage. It is probable that most cases of epileptic mania are really examples of post-epileptic automatism. One of my patients, immediately after an attack, struck a friend who was with him a violent blow on the face, mistaking him for another person, and was in consequence taken to a police station under the suspicion of being drunk. Another patient, immediately after a fit, threw her baby downstairs. Without doubt, many crimes have been committed in this state. The possible relation of criminal actions to epilepsy has formed a medico-legal question at many criminal trials.

The emotion displayed is sometimes of a more cheerful character. One patient always laughed and sang for a few minutes after every attack of *petit mal*, and in another, a girl of twenty, each slight seizure was followed by a paroxysm of kissing; she would kiss every person near her, irrespective of sex, and even seize her pillow and kiss it most affectionately.

In some cases there is considerable uniformity in the character of the actions which are performed in this post-epileptic state. A woman, for instance, aged 45, had been the subject of epilepsy since six years old; severe attacks, with tongue-biting, occurred every few months, and slight attacks, consisting of the epigastric sensation and loss of consciousness, much more frequently. From the first, after both kinds of fits, she commonly tore her hair. When quite a little girl she was once found unconscious in a room by herself, and had torn a quantity of hair out of her head.

*Post-Epileptic Hysteria.*—Instead of presenting automatic action of the character described, some patients pass, on the cessation of an epileptic fit, into a state of hysteroid convulsion. This sequel is met with only in patients who are of the age at which hysteria is common, namely, under thirty-five, and although common in both boys and girls, it is comparatively rare in young men, and is frequent in young women. Some patients always present it after their attacks, others never do. Hence it is evidently the result of two causes. The first is the preceding cerebral 'discharge,' leading to insubordinate action of lower centres, perhaps in consequence of the mere loss of control of the highest. Secondly, it is the result of some preceding morbid state of the brain, such as leads to the manifestations of hysteria, apart from epilepsy. It is the possession of this special instability which leads to the occurrence of the hysteroid convulsion under the circumstances. Other patients, without this special predisposition, have quite similar attacks, but without any hysteroid sequelæ. In some of the patients who present these symptoms there are, at other times, various manifestations of the hysterical temperament; in others these are absent, except after the epileptic seizures. In the latter the hysterical condition is apparently not sufficiently developed to lead to independent symptoms, although it manifests itself during the morbid state immediately after an epileptic fit. Hence we must regard the patients who present this sequel as really the subjects of both epilepsy and hysteria. Some of them have only attacks of *petit mal,* and in such cases it may be extremely difficult to say whether the hysteroid attack is simple, exists alone, or whether it is preceded by an attack of minor epilepsy, and is really a post-epileptic phenomenon. On the other hand it is very common for these post-epileptic hysteroid convulsions to be mistaken for major epileptic seizures. Patients are often said to have frequent attacks of *petit mal*, and occasionally severe fits, or to have been liable to slight attacks for months or years, and then to become subject to severe attacks, when the latter are found, on minute enquiry or actual observation, to be merely hysteroid convulsion occurring after the attack of *petit mal.*

The occurrence of such convulsion as an occasional sequel to severe attacks of epilepsy has been already mentioned. It is, however, far more frequent after minor seizures. But it will be convenient to postpone the description of these attacks until the character of hysteroid seizures is considered in detail.

There is one post-epileptic action which is automatic, occurring alone and also as part of hysteroid convulsion, and which is of great importance on account of its danger. It is the tendency to turn over on to the face. When the patient is in bed, suffocation may easily occur in this posture, and without doubt many epileptics have died from this cause. Some patients never present this tendency; others always do, and their friends should be made aware of its danger. The peril is the greater, because the action is the result of the state which causes hysteroid convulsion, and patients who present it are therefore often thought to suffer merely from hysterical fits. It was illustrated, only too clearly, by the case of a girl, aged fifteen, who had suffered from 'fits' for a year. There was strong hereditary tendency. Her own father and brother were insane. Her mother's father and brother were both epileptic and insane. Her fits occurred daily, both sleeping and waking. The history of epileptoid seizures was clear, but after the fit was over she would turn on to her stomach and then kick and struggle, sometimes for hours. In the hospital, however, she had no distinct epileptic attacks, and the attendants were inclined to regard her attacks as purely hysterical. She sometimes fell down on her back and lay quiet without change of colour, and could then be brought to by stopping the mouth and nose for a few seconds. In other attacks she would fall suddenly and then clasp her hands tightly, stretch herself out stiff and 'work her eyes about,' and always turn on to her face and try to burrow her face into the pillow or against the floor. From this state she could be quickly roused by water. One night she was noticed to be all right at 3 A.M. At 6 o'clock the nurse went to her bed to call her, and found her lying on her face, with the face against the pillow, and dead. No one in the ward had heard a sound.

## MENTAL DISTURBANCE IN EPILEPTICS.

*Paroxysmal Mental Disturbance; Epileptic Mania.*—The subjects of epilepsy sometimes, but rarely, present sudden paroxysmal outbursts of mental derangement, often with violence, and a tendency to injure others. The maniacal attack is usually brief, often lasting for a few minutes only, sometimes for an hour or two. It was formerly thought that such outbursts represented and constituted an attack of epilepsy. In the majority of cases, however, it is certain that, as Hughlings Jackson maintains,[1] the mental derangement is the sequel to a fit, and is really a form of the automatic action which may succeed an attack, sometimes severe, much more commonly slight.

If the preceding minor attack is very slight, it may readily be overlooked, and hence the psychical disturbance be regarded as primary. Whether this is true of all instances of maniacal outbursts in connection with epilepsy is doubtful. In rare cases (as Esquirol and other good observers have pointed out) an act of violence precedes a fit. We have seen, in the account of modes of onset, that the aura of an attack may sometimes consist in a complex or coordinated mental process, or a visual idea. There is nothing inconsistent with ascertained facts in a sudden act of violence as the result of the initial discharge. One of my patients always hopped round the room before he fell in a fit. Cases in which a patient suddenly runs, or turns round and walks backwards, before a fit, have been mentioned, and a similar cerebral process to that which causes such a proceeding may declare itself in an act of violence. It thus seems possible that a maniacal outburst may be the only effect of a 'discharge,' may alone constitute an attack. Such cases are certainly rare, but it is doubtful whether we are justified in denying their occurrence.

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[1] *Med. Press and Circular*, Nov. 11, 1874, p. 409.

Occasionally, after a fit, or, more frequently, after a series of fits, an attack of mental disturbance may come on which lasts for several days. It may be simply a demented state, or there may be hallucinations, with irritability and even violence. In the latter cases, according to Voisin, there may be an elevation of temperature to 102° or 104°. This state sometimes comes on in an epileptic patient without immediate association with attacks, and even during the temporary cessation of attacks. It is not uncommon for a patient's mental state to be worse, his temper more irritable, during the temporary cessation of severe fits, and this apart from the occurrence of slighter seizures, and in rare cases the disturbance amounts to actual mental aberration.

*Interparoxysmal Mental State.*—The mental state of epileptics, as is well known, frequently presents deterioration, and this constitutes one of the consequences of the disease which is much dreaded, and is often most serious. In its slighter form there is merely defective memory, especially for recent acquisitions. In more severe degree there is greater imperfection of intellectual power, weakened capacity for attention, and often defective moral control. Mischievous restlessness and irritability in childhood may develope to vicious and even criminal tendencies in adult life. Every grade of intellectual defect may be met with, down to actual imbecility.

The mental state must not be regarded, in all cases, as entirely the effect of the disease. It is certainly, in some, the expression of a cerebral imperfection of which the epilepsy is another manifestation. In such instances mental defect exists before the occurrence of the first fit. In other cases, however, which constitute a majority of the whole, the failure must be regarded as a consequence of the disease. It distinctly succeeds the fits in point of time, and may lessen very much when the fits are arrested by treatment. It is not surprising, therefore, that in cases in which a mental defect exists before the fits commenced, this should be greatly intensified by the subsequent attacks. It is among these cases that many of the worst forms of mental failure are met with.

The conditions with which the occurrence of mental defect is associated have been carefully compared in sixty-four cases in which considerable mental defect existed, and of which the history could be ascertained.

It is doubtful whether *sex* influences its occurrence; females constituted 58 and males 42 per cent. This is a rather greater preponderance of females than obtained in the whole series (53·4 per cent.), but the difference is scarcely sufficient to be of significance, on account of the small number of cases. As far as the facts go, they tend to show that females are rather more likely to suffer from mental defect than males. A similar conclusion was reached by Reynolds.[1] The proportion of cases with *inherited tendency* was 29 per cent. as against 36 per cent. in the whole number. From this it may certainly be inferred that heredity does not increase the probability of mental failure.

The *age* at which the disease commences has, however, a very distinct influence. In the following table the percentage proportion of the sixty-four cases with considerable mental defect, and of the whole series (1450 cases), commencing at each period of life, are compared:—

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[1] *Loc. cit.* p. 169.

| | With mental defect | Whole series |
|---|---|---|
| Under 10 | 55 per cent. | 29 per cent. |
| 10–19 | 36 „ | 46 „ |
| 20–30 | 3 „ | 15·7 „ |
| Over 30 | 6 „ | 9·3 „ |

Thus the tendency to mental failure is, as Romberg insisted (although with too great emphasis), greatest in the cases which commence in childhood, under ten years, and is much less in those which commence between twenty and thirty. It is also greater in early than in later childhood, the first five years yielding no less than 39 per cent. of these cases, and only 18 per cent. of the whole series. This, doubtless, depends upon two causes. During early life the brain suffers in great degree from any unfavourable influences, and, secondly, many cases of cerebral defect, congenital, or the result of damage during birth, become epileptic during the first five years of life. Mental failure is, however, occasionally met with in cases of epilepsy which commence during the second half of life.

The *duration* of the disease has also a distinct influence on the occurrence of mental failure. The disease had existed for several years in a larger proportion of the cases which presented mental failure when they came under observation, than of those which presented no conspicuous failure. This is clearly shown by a comparison of the duration of the disease in fifty-six cases with considerable mental failure, and in a thousand cases in which no mental failure was noted, and in which therefore it was certainly either slight or absent. The percentage proportion of each series which had lasted for the time stated is given in the following table:—

| Duration | Mental failure conspicuous (56 cases) | | Mental failure slight or absent (1000 cases) | |
|---|---|---|---|---|
| Not exceeding 1 year | 12·7 | 27·2 | 25·2 | 55·1 |
| 2–4 years | 14·5 | | 29·9 | |
| 5–9 „ | 36·4 | 72·8 | 21·5 | 44·9 |
| 10 years and over | 36·4 | | 23·4 | |

Thus, of the cases with mental failure, nearly three-quarters had lasted more than four years, while less than half the cases without mental failure were of this duration. The influence of duration is better seen thus than by comparing the mean duration of the two sets of cases, which was nearly the same: that of the thousand cases without mental defect was six years; that of sixty cases with mental defect was seven years. The slight difference between the average duration of the two sets of cases is due to the fact that cases of very long duration are much more common without than with mental defect. Thus no case with considerable mental defect had lasted for more than twenty-one years, while the series of cases without mental change included no less than thirty-seven cases of longer duration than twenty-one years, and nine cases of longer duration than thirty years; in one the disease had existed for fifty-four years. The reason for this probably is that the cases of very long duration with much mental change usually find their way into asylums.

The long duration of some of the cases without mental defect illustrates the fact, sufficiently clear from the table given above, that the intellectual change is not a simple effect of the duration of the disease, which is one, but not the only, and probably not the chief, factor in its causation.

*Frequency of Attacks.*—An examination of the influence of frequency of attacks shows that it cannot be regarded as possessing a considerable influence. The attacks occurred with sufficient regularity to allow their frequency to be estimated in only forty-four of the cases with considerable mental defect; and in the following table these are compared with 522 cases in which no mental defect was noted:—

| Interval | Cases with mental failure (44) | Without mental failure (522) |
|---|---|---|
| Not exceeding 1 week . . | 50 per cent. | 47·2 per cent. |
| 1 week to 1 month . . . | 39 „ | 34·4 „ |
| Over a month . . . . | 11 „ | 18·4 „ |

The chief difference between the two sets of cases is the comparative rarity of long intervals in cases with mental failure. In only one of the forty-four cases did the interval between the fits exceed two months, whereas in the series without mental defect this interval was exceeded in fifty-three cases. The average interval in the latter series (without mental failure) was twenty-six days, whereas in the cases with mental defect it was only fifteen days. But the fact that in the two series the proportion of cases in which the intervals were very short is so nearly the same, shows that mere frequency of attack can scarcely be regarded, by itself, as a cause of mental failure.

The influence of *form of attack* on mental failure is a subject on which great diversity of opinion exists. Most authorities are of opinion that minor attacks, in which there is loss of consciousness and little more, exert a more prejudicial influence than the severe fits. It is certain that the gravest forms of mental defect are sometimes met with in cases in which there are only minor attacks, but they also occur in cases in which the attacks are severe and *petit mal* is absent. On the other hand, that minor fits, even when frequent and occurring for a long period, do not necessarily cause mental failure, is certain. One of my patients, a girl of twenty-five, had suffered for twenty years from *petit mal,* with complete loss of consciousness; thirty to forty attacks occurred daily, and yet there was no defect of mental power—not even failure of memory. Hence it would seem that no necessary, and perhaps no considerable, influence is exerted by mere form of attack, so far as the distinction between slight and severe fits is concerned. That there are varieties of these two forms which exert a more prejudicial influence than others, is highly probable, but evidence on the point is not yet forthcoming.

The conclusion from these considerations is that mental failure is determined less by single conditions than by their combinations, and that it is probable that a more potent cause than the attacks themselves consists in a predisposition to suffer under their influence—a predisposition which is related to the ultimate causes of the disease rather than to its developed characters. Of the latter, early age at commencement, long duration of the disease, and frequency of attack are more influential than the sex of the sufferer, the existence of heredity, or even the character of the attacks, so far as concerns the mere distinction between major and minor fits.

## GENERAL HEALTH OF EPILEPTICS.

So far as can be ascertained, many epileptic patients are in perfect and even robust health. The various organic functions, in the intervals of the paroxysms, are normal. More frequently, however, the general health exhibits some imperfection. The digestive organs are often deranged, the bowels are constipated, flatulence is troublesome, the appetite bad, and the tongue is furred. In some patients there is a voracious appetite, especially after attacks. Often the disturbance is increased by the long-continued administration of bromides. The circulation is feeble, the pulse small, unduly frequent, and very often slightly irregular. The heart's action is excited, and slight dilatation of the left ventricle can frequently be recognised. Valvular lesions are not rare, and will be subsequently considered. 'Nervous urine,' pale and of low specific gravity, is frequently passed. The constitution of the urine is usually normal.

# CHAPTER V.

## *EPILEPSY AFTER HEMIPLEGIA.*

MANY organic diseases of the brain not only cause convulsions during their active stage, but, when their active stage is over, are followed by epileptiform attacks which may recur for many years, sometimes during the whole of life. Such convulsions, for instance, are not uncommon as a sequel to hemiplegia which was sudden in its onset, and therefore due to a vascular lesion. These cases of 'post-hemiplegic epilepsy' are of considerable practical importance, and constitute a considerable proportion of the cases of chronic convulsive disease.

*Etiology.*—Hemiplegia, occurring at any period of life, may be followed by recurring convulsions; but this sequel is far more frequent in the cases in which the onset of the paralysis is during infancy, than in the cases of hemiplegia in adult life, as is shown by the following table of sixty-six cases of which I have notes:—

| Onset | Males | Females | Total |
|---|---|---|---|
| Under 5 | 16 | 28 | 44 |
| 6–10 | 3 | 3 | 6 |
| 11–20 | 1 | 4 | 5 |
| 21–40 | 3 | 2 | 5 |
| Over 40 | 4 | 2 | 6 |
| | 27 | 39 | 66 |

It will be seen that in two-thirds of the cases the hemiplegia came on under five years of age. In nearly half the cases (ten males and eighteen females) the onset was before the end of the second year. In only four cases did it actually date from birth. Females suffer from post-hemiplegic epilepsy more frequently than males, but this depends especially on the larger number of girls than boys who become affected under five years, the former being nearly twice as numerous as the latter. In the smaller number of cases in which the hemiplegia comes on after twenty, males preponderate.

It is more common for the hemiplegia, in these cases, to be on the left side than on the right, but this statement is only true of the cases in which the affection comes on in early childhood. The facts, in regard to sixty-two cases, are as follows:—

| | Right | Left | Total |
|---|---|---|---|
| Under 5 | 16 | 26 | 42 |
| 6–10 | 3 | 3 | 6 |
| 11–20 | 2 | 2 | 4 |
| Over 20 | 5 | 5 | 10 |
| | 26 | 36 | 62 |

Thus, in the cases in which the hemiplegia dates from the first five years of life, it is much more common for it to be on the left side; but when it comes on after the fifth year it is as frequently on one side as on the other.

The cause of the hemiplegia in these cases can only be inferred from the conditions of its onset. It is known, however, that in young children sudden hemiplegia is almost always the result of spontaneous thrombosis in a cerebral vessel. In the majority of cases no exciting cause could be ascertained. In a few the onset was during an acute illness, during measles in five, during convalescence from scarlet fever in three, during mumps in one; one was in the subject of inherited syphilis, two had had falls on the head. All these are conditions in which vascular obstruction is known to occur. The history of many of the cases which occurred after childhood points to a similar conclusion. Two were the subjects of constitutional syphilis, in two there was heart disease, in one the onset was during rheumatic fever, in one after labour. Thus in a very large number of cases there was reason to believe that the lesion was vascular obstruction. The same opinion has been expressed by Hughlings Jackson.[1] It is well known that in softening from this cause the brain tissue adjacent to the softened area is usually damaged by the collateral congestion, which leads to abundant punctiform extravasation; if grey matter is thus damaged, it is easy to understand that it may recover imperfectly, and that a permanent condition of instability may result. Moreover, thrombosis in veins (the most common lesion in childhood) often affects the surface of the brain, where organic disease most frequently causes convulsions. In two cases the onset was during an acute cerebral illness, possibly meningitis.

In some of the cases which dated from birth, labour had been difficult, and it is probable that injury to the surface of the brain occurred.

In most cases no other cause for the convulsions could be traced than the damage to the brain. In a few, however, there was an inherited tendency to epilepsy. Two adults were the subjects of lead-poisoning and albuminuria; the former is known to be capable alone of causing convulsions, and may have produced a predisposition to their occurrence.

*Symptoms: Onset.*—In half the cases (34) convulsions attended the onset of the hemiplegia, which was, in 29 of these, in early childhood. These initial convulsions were usually severe and repeated. Sometimes two or three attacks occurred, increasing in severity, and after the last the child was found to be paralysed. In other cases the convulsions recurred during several days, and even a week.

The chronic convulsions which succeed the hemiplegia date from its onset in one half of the cases. A few days or weeks after the establishment of the paralysis the patient has another fit, soon afterwards another, and so on until the convulsive disease is established. In other cases there is an interval after the onset of the paralysis before the attacks occur. The patient may be well, except for the remains of the hemiplegia, and after a few months or years may become subject to fits. In about a sixth of the cases the interval is more than five years, and occasionally it is as long as fifteen or twenty years. The intervals in 33 cases are shown in the following table:—

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[1] *Brain*, April 1881, p. 437.

| | | |
|---|---|---|
| Fits from the onset of the hemiplegia | | 14 |
| Interval less than a year | 2 | |
| „ 1–2 years | 5 | |
| „ 2–5 „ | 3 | |
| „ 5–10 „ | 4 | |
| „ 10–20 „ | 5 | |
| Total in which there was an interval | | 19 |
| | | 33 |

Thus, if the fits do not recur within a few days, there is usually an interval of more than a year.

In some of the cases in which an interval elapses, the recurrence of convulsive attacks may be traced to some exciting cause. In one patient, for instance, the hemiplegia came on without apparent cause at one year old; the onset was attended by convulsions, but no other fits occurred until two, during an attack of whooping-cough. In another case the hemiplegia came on without convulsions at four months, but fits occurred during the first dentition, and again during the second dentition at seven, and after the latter they were permanent. Again, one patient had hemiplegia, after a fall on the head, at two years of age, with convulsions at the onset, but no other fits until seven years of age, when the attacks recurred after another fall on the head, and continued until the patient came under treatment at eleven. It is not very rare, in cases in which the hemiplegia comes on during the first years of life, for the patient to be free until puberty, and then, at thirteen, fourteen, fifteen, or sixteen, the fits commence and continue. No doubt, in these cases, their occurrence is due to the influences which determine the frequent commencement of idiopathic epilepsy at this period.

The only relation ascertainable between the occurrence of convulsion at the onset of the hemiplegia and the interval which elapses before subsequent fits, is that in all the cases in which the attacks are continuous from the onset, this was accompanied by convulsion. The converse, however, is not true. In many cases in which convulsion occurred at the onset, an interval of several years elapsed before the fits recurred.

In the cases in which convulsions succeed hemiplegia which comes on late in life, there is rarely any long interval of freedom after the onset of the hemiplegia. Usually in the course of a few months, or at most of a year, the seizures occur.

*The condition of the paralysed limbs* in these cases varies. In most cases a considerable degree of paralysis persists, especially in the arm. That in the face is usually slight, and in the tongue often absent. The leg has usually recovered almost completely, as is the rule in hemiplegia occurring in early life. The weakness in the arm is, in most cases, distinct, and often great; sometimes the hand remains completely paralysed, a little power in the shoulder and elbow alone remaining. In other cases recovery is almost perfect; the patient may be unconscious of any persistent weakness, although usually slightly defective power in the arm or face may be found if carefully searched for. In very rare cases no trace can be found of any weakness, although the initial hemiplegia may have been well marked, and the convulsions may affect that side only.

When the hemiplegia comes on in early life, the paralysed limbs are usually smaller than those on the other side, and smaller not only in circumference but in length, in consequence of a retardation in the growth of the bones, in which some of the trunk bones, as the scapula, may participate. The arm and leg may each be one or two inches shorter than that of the other side. The arm may be the seat of late rigidity. The hand is often affected by a condition which I have termed 'mobile spasm,' and which has been called 'post-hemiplegic chorea' by

Weir Mitchell and Charcot—spasm which causes slow spontaneous movements of the fingers and thumb, and incoordination of voluntary movement. In an account of this form of post-hemiplegic spasm I pointed out, some years ago,[1] the frequency with which it is associated with convulsions, and the association is equally conspicuous if these cases are studied from the side of convulsion. Such spasm is found in one-half of the cases of post-hemiplegic epilepsy. Of the 66 cases, it was present in 34. Its character and degree vary. The chief feature is the tendency to spasm of the interossei, whereby the fingers become flexed at the metacarpo-phalangeal joints and extended at the middle and distal articulations. Frequently the continued extension at these joints leads to over-extension and subluxation, so that the round heads of the first phalanges project unduly on the palmar aspects of the fingers. This form of spasm presents a striking contrast to that of 'late rigidity,' in which the spasmodic contraction chiefly affects the long flexor of the fingers, which are thus flexed at the middle and distal joints. The thumb may be bent in to the palm, or pressed against the first finger, or over-extended. Often the wrist is strongly flexed by the contraction of the special carpal flexors. In other cases, or at other times, the wrist becomes strongly extended. When the mobile spasm is slow, considerable and spontaneous, the hand presents the condition known, after Hammond, as 'athetosis.' In other cases the spasm may be slight and inconspicuous when the hand is at rest, but may be distinct on movement, causing a slow incoordination, the result of the irregular spasm, in which the overaction of the interossei is often conspicuous. There may also be incoordination, but rarely spontaneous movement, at the elbow and shoulder joints. The muscles may be of good size, and are sometimes even larger than on the healthy side, in consequence of the constant spasmodic exercise, and this even although the limb is shorter than the other. A slight tendency to similar overaction may be sometimes noted in the side of the face. In the foot it is often distinct, causing a tendency to inversion of the foot and over-extension of the toes.

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[1] *Med. Chir. Trans.* 1876.

This mobile spasm is a frequent result of infantile hemiplegia, but its association with convulsions is not confined to cases of infantile onset. Mobile spasm was conspicuous in cases of convulsion in which the hemiplegia came on at seven, ten, eleven, twenty-one, and twenty-seven years, although there was no mobile spasm in the cases in which the hemiplegia came on after thirty.

The association of the two symptoms is too frequent to be accidental, and they are probably the result of the same morbid state. In the paper above alluded to I pointed out that in most cases of mobile spasm after hemiplegia there is evidence that the lesion was vascular obstruction and not hæmorrhage, and I suggested, as its probable cause, the damage to adjacent motor grey matter, which recovered imperfectly, and remained in a state of instability, with lessened resistance, overacting spontaneously, or on the excitation of a volitional impulse. We have just seen that the conditions of onset of the cases of hemiplegia which are followed by convulsions point to the same conclusion—that the initial lesion is vascular obstruction, and that the convulsions are the result of the imperfect recovery of damaged grey matter in the vicinity of the lesion.

It is of interest also to note that the common posture of the hand in a case of post-hemiplegic mobile spasm, pronation, strong flexion of the wrist, interosseal position of the fingers, is precisely that which is produced by the spasm of an ordinary epileptic fit.

Sensation on the paralysed side is usually normal. In a few cases I have found a slight persistent defect of sensibility to touch. In no case was there any affection of the special senses.

*The convulsive attacks* vary much in character. In a few cases the convulsion is general from the first, and similar to the attacks in idiopathic epilepsy. In the majority of cases the convulsion begins in, or is confined to, the paralysed side. This was the case in 34 out of 50 patients in whom the character of the convulsion could be definitely ascertained. In many the convulsion affects all parts of the paralysed side, and does not spread to the other; in others, the unparalysed side is involved secondarily. In a few only part of the paralysed side is involved, usually the arm, or arm and side of the face; less commonly it is confined to the leg.

A distinct warning is present in a much larger proportion of cases of post-hemiplegic epilepsy than of the idiopathic form. Of 38 cases in which the presence or absence of a warning was noted, it was constantly absent in only five.

The frequency with which the several forms were met with, in cases of right and left hemiplegia respectively, is shown in the following table:—

| | Side hemiplegic | | |
|---|---|---|---|
| Commencement | Right | Left | Total |
| Hand | 1 | 6 | 7 |
| Arm | 2 | 1 | 3 |
| Face | — | 1 | 1 |
| Head, turning | 1 | — | 1 |
| Leg | 1 | 4 | 5 |
| Arm and leg | 1 | — | 1 |
| Both feet | 1 | — | 1 |
| Ocular | 1 | 1 | 2 |
| Malaise | 1 | 1 | 2 |
| Vertigo | 1 | 1 | 2 |
| Headache | — | 1 | 1 |
| Vomiting | — | 1 | 1 |
| Epigastric | 1 | 3 | 4 |
| Throat | 1 | 1 | 2 |
| | | | 33 |
| No warning | 3 | 2 | 5 |

As might be anticipated, the convulsion commences in one of the paralysed limbs in a considerable proportion of cases, and this deliberate commencement constituted the warning in 17 out of 32 cases in which a warning was present. The commencement was most frequently in the upper limb (in ten cases), and it was in the hand in seven, most frequently by a sensation, which in several passed up the arm and down the side. In a minority of cases the commencement was by distinct spasm in the hand (twitching of the fingers, twitching of the thumb, and closure of the hand). Much less frequently (in three cases only) the warning was in the arm as a whole, and in only one of these was it spasm—a sudden flexion of the arm.

Much less commonly the commencing fit is first felt in the lower limb (five cases), and in all the initial symptom was in the foot, usually a sensation in the toes, less commonly spasm. The sensation usually passed up the leg and side and down the arm: the spasm passed up the side, and then attacked the arm without any deliberate descent of the limb. In one case the warning was 'pins and needles,' felt simultaneously in both arm and leg. Commencement in the face is much less common, and was noted in only one case—twitching of the left side of the face with inability to speak. In one case the initial symptom was rotation of the head.

In all the cases commencing in this manner the convulsion was confined to, or involved first, the paralysed side; in a large number the unparalysed side escaped altogether. In the case in which the fit began in the face, and in one in which it commenced in the hand, it did not extend beyond the arm.

Fits commencing in the hand or the foot appear to be much more frequent as a sequel of right than of left hemiplegia. Of six out of seven cases in which the attacks began in the hand, and in four out of five cases in which the commencement was in the foot, the hemiplegia and convulsions were on the left side. On the other hand, in the cases in which the convulsion began in the upper part of the arm and in the face, the affection was almost as frequent on one side as on the other.

Ocular warnings are much less common in these cases than in idiopathic epilepsy. In one only was there an initial visual sensation, 'a fire before the eyes, going to the left,' and the fit was left-sided. In one there was an ocular sensation, 'as if the eyes were riveted together,' followed by right-sided convulsion. A vague feeling of illness, vertigo, headache, vomiting, and a sensation of choking, are rarer warnings. An epigastric aura is rather more frequent, and may occur in cases in which the convulsion is unilateral. In one patient the attacks at first commenced deliberately in the hand, but afterwards by the epigastric-throat aura only.

The convulsion in these cases usually consists of both tonic and clonic spasm, and is of the same character as in ordinary epilepsy. Rarely it is clonic only. Consciousness is usually lost; sometimes, during a slight attack, it is unaffected.

After a fit is over, the state of the patient is similar to that after attacks of idiopathic epilepsy, but temporary weakness, 'post-epileptic paralysis,' is especially common. In two cases temporary deafness succeeded the fits.

Minor attacks are frequent in cases of post-hemiplegic epilepsy, and usually consist in the aura of a severe fit, often without loss of consciousness. In a few cases, instead of any spasm, an initial sensation in the limbs is followed by transient powerlessness, apparently the result of inhibition (see p. 102). Occasionally attacks of *petit mal* occur which are quite similar to those of the idiopathic form—a sudden faint or sudden start, sometimes accompanied by an epigastric sensation.

Hysteroid seizures are not at all rare in these patients. They may succeed epileptoid attacks, major or minor, or may occur alone. Some of the most severe hysteroid fits I have seen were in a girl the subject of infantile hemiplegia, whose arm was permanently paralysed. The attacks came on without any indication of initial epileptoid seizure, and I have known them to continue, unless interfered with, for several hours—violent opisthotonos, bounding movements, biting, tearing of hair, etc. The same patient had also other hysterical manifestations, attacks of violent laryngeal spasm, rapid breathing, and a phantom tumour. But she had also severe epileptic fits beginning on the paralysed side, one of which I saw.

In the cases in which the hemiplegia comes on early in life, mental defect is very common. The development of the whole brain seems to be retarded by the lesion, and the intellectual power is permanently below par. The patients are often mischievous and troublesome during childhood, and subsequently vicious, and often immoral.

# CHAPTER VI.

## *HYSTEROID OR COORDINATED CONVULSIONS. —HYSTERO-EPILEPSY.*

### GENERAL CHARACTERS OF ATTACKS.[1]

THE characteristic of the convulsions which are called 'hysterical,' 'hysteroid,' 'hystero-epileptic,' or 'hysteria major,' is, as stated in the introductory chapter, that the spasmodic movements are of a more or less coordinated character. The grouping of muscular contractions is, in the main, such as may be produced by the will. Hence the spasm has a somewhat purposive aspect. Rigid fixation of the trunk and limbs alternates with wild movements, in which the limbs are thrown about; the arms strike out, the legs kick, the head is dashed from side to side. In the rigid spasm the back may be arched, so that the patient rests on the head and the heels, or the trunk may be straight, and the limbs extended, less commonly flexed. The tonic spasm may be broken by quick quivering movement, a form of clonic spasm, but unlike the clonic spasm which is characteristic of epilepsy, although a somewhat similar form of spasm is occasionally seen in epileptic fits (see p. 78). Sometimes the spasm is rather of the character of rapid, rhythmical, quasi-volitional movements than of actual clonic character. Laryngeal spasm, in some cases, forms a conspicuous feature of the attack. These phenomena are interrupted by periods of comparative tranquillity, attended, in many patients, by delirium, and sometimes by manifestations of emotion, usually of fear, which become more and more intense until they culminate in a paroxysm of convulsion.

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[1] The following description of the symptoms of these attacks is based upon cases which have come under my observation, some of which are described in detail. Since the disease, as met with in this country, differs in some respects from the characters which it presents in individuals of other races, and since the description of the attacks by Charcot has been the chief source of information available for recent writers, the account here given differs, in many respects, from that which has lately become current; but it will, I think, be found an accurate description of the phenomena of these cases as they are met with here.

At the onset of one of these attacks there may or may not be a warning sensation. The patient usually falls to the ground, but rarely with the violence with which an epileptic falls. Very often the fall is gradual—a sliding down rather than a fall. After falling the violent coordinated movements may commence instantly, as soon as the body touches the ground. More commonly there is an initial period of tonic rigidity, sometimes succeeded or accompanied by the quick clonic spasm above described, before the coordinated movements come on.

Consciousness is rather changed or impaired than lost during these attacks. A sort of automatic consciousness, if the expression may be allowed, is present, but not the highest consciousness, which is necessary for the subsequent reproduction of the mental states which arise during the attack. After a fit is over, therefore, the patient remembers nothing about it,—may be quite unaware that it has occurred. In many cases the patient appears to be actually unconscious during the paroxysms of violence, but in the intervals appears rational, although peculiarities in tone and manner often tell those who know the patient that the end has not yet come, and presently a fresh paroxysm occurs. The duration of the attacks varies from a few minutes to several hours, unless they are cut short by treatment. At the end the patient usually goes to sleep for a few minutes and wakes up well; or the attack may suddenly cease without sleep.

These attacks vary greatly in severity and character. When slight they are of the trifling character popularly known as a 'fit of hysterics,' into which an emotional patient will 'work herself up,' and in which there is no distinct affection of consciousness. When severe, the violence of the spasmodic movements is almost inconceivable. The head may be dashed against the floor with great force, and the body be alternately bent forwards and then thrown back in opisthotonos, so that the patient almost bounds off the bed. The more restraint is used, the more is needed, and these are the attacks in which the friends of a patient will tell us that 'six persons had to sit on him to keep him down.'

Similar variations are seen in the mental disturbance which attends the attack. This may be trifling, and amount only to an abnormal emotional state, or it may be so severe that for a time the patient is in a state of maniacal frenzy. Between these extremes every variety of mental disturbance may be met with. Some patients will scream loudly throughout an attack. Others will talk in an unnatural manner. One girl always counted backwards with extreme rapidity. In some patients a curious condition is observed, which may be termed therio-mimicry. The noises and actions of animals are strangely imitated. The patient mews like a cat, or, much more commonly, barks like a dog. Still more frequent is a tendency to bite. Patients sometimes, but rarely, bite themselves. I have known the lip to be bitten, and two patients bit their own fingers, in one case so severely as to leave a permanent scar an inch long. Very commonly, however, the patients try to bite other persons, and the tendency renders considerable care necessary on the part of the attendants. Not only do they bite, but they do so in a curiously animal manner. A case is described in a subsequent page in which a lad of sixteen, after failing in an attempt to bite an attendant's hand, seized the corner of his pillow between his teeth, and, throwing his head back, shook the pillow, just as a dog shakes a rat, or as the lion shook Livingstone. This therio-mimicry may be in part truly mimetic, but it seems to be, in part, the manifestation of some strange animal instinct which we possess in a latent or modified condition, like our canine teeth. A lad of twelve was found, in one attack, in the act of 'worrying' a cat, having seized it by its neck with his teeth. The cat was making a vigorous defence with its claws, but was nearly killed before it could be extricated.

Such hysteroid convulsions may, and often do, occur alone, the patient presenting, in the attacks, no other phenomena, and having no other kind of attack. But they also occur, with considerable frequency, in epileptic patients after genuine epileptic seizures. When the epileptic fit is over, the patient passes into this condition of hysteroid or coordinated convulsion, with alternating delirium. The condition lasts for a variable time, a few minutes to an hour or more. These post-epileptic paroxysms may occur after severe attacks, as already mentioned. But they are also common after minor seizures. A patient may, for instance, have attacks of distinct epileptic character, slight or moderate, preceded by an aura, and attended with loss of consciousness or with slight characteristic convulsion. Then, sometimes after such attacks have occurred alone for years, each of them, or some of them, are followed by hysteroid convulsion. The epileptic attacks are of the same character as before, but after each, instead of simple sleep or quick recovery of consciousness, the patient presents the coordinated movements, throwing about of limbs, opisthotonos, and delirium, which have been described as characterising the simple hysteroid seizures. The relation of the two will be considered in the next chapter.

The cases of simple hysteroid fits, with a period of initial fixed tonic spasm, bear close resemblance to the cases in which hysteroid convulsion succeeds a slight attack of genuine epilepsy. It may be asked, are not the two really identical? Are not all cases which present this sequence either cases of pure hysteroid attacks, with initial symptoms apparently, but not really, epileptic; or are they not all cases of slight epileptic seizures, succeeded by hysteroid convulsion as a post-epileptic phenomenon?

That many of these cannot be regarded as simple hysteroid fits is proved by the fact that the patients have, or have had, other attacks which are distinctly epileptic seizures, without any hysteroid symptoms, but identical with those which precede the coordinated convulsion, and further that the pure epileptic attacks, in some of these cases, are preceded by a warning similar to that which heralds the compound fit.

On the other hand, that the cases in which there is an initial period of stillness or tonic rigidity are not all instances of true epilepsy followed by hysteroid convulsion, is proved by the facts that many of the patients never present any isolated or more distinct epileptoid seizures; that the attacks rarely occur when the patients are alone; that the liability to them, their occurrence, and their cessation, are all influenced by the patient's psychical condition; and that they are arrested by the treatment for hysteria, and are not influenced, or only to a very slight degree, by the treatment suited to epilepsy. Further, the initial pseudo-epileptic phenomena may recur repeatedly during the progress of the fit, affording thus a demonstration that they are actually part of the hysteroid attack.

It has been mentioned that, in the attacks which occur in the natives of this country, the initial tonic stage (although it may resemble that seen in certain aberrant forms of epilepsy) bears little resemblance to the spasm of a typical epileptic fit. There is not the irregular strained fixation of limbs, and deviation of head and eyes, which occur at the onset of the epileptic seizure, nor is there the violent shock-like clonic spasm, commencing gradually, and diminishing in frequency while it maintains its force. But in individuals of some other races, and especially in the French patients whose cases have been so carefully studied at the Salpêtrière, the disease attains a greater degree of intensity than in this country. In them, as is shown by the description of the disease given by Charcot, and especially by Richer in his recent admirable work on the disease,[1] the attacks commence by a convulsive seizure resembling a true epileptic fit very closely. The patient suddenly falls unconscious, in general, severe, tonic spasm, with deviation of the head, and the tonic spasm passes gradually into clonic spasm, succeeded for a few seconds by coma and stertor. This constitutes the first or 'epileptic' stage. Then violent coordinated spasm comes on, the stage of '*grands mouvements*,' opisthotonos, bounding movements, etc., such as have been described above, but of extreme violence. This is succeeded by a period of mental and emotional disturbance, with manifestations of joy, anger, or erotism—the stage of delirium. Sometimes there is a final stage of terrifying visions.

It may be asked, is not this merely the sequence already described—a true epileptic fit succeeded by post-epileptic hysteroid phenomena? Charcot and Richer have pointed out that, close as is the resemblance of the first stage to an epileptic fit, there are certain important distinctions which indicate an essential difference. (1) The attack is often preceded by a peculiar mental state, with hallucinations, and frequently accompanied by transient contractures of one or another limb. This condition gradually increases in intensity until the attack comes on. (2) The tonic spasm with which the epileptoid stage commences is usually immediately preceded by violent movements of the limbs. (3) An attack may be brought on by compressing the ovaries, or in some cases by touching certain 'hysterogenic points' on the surface. (4) At any period of the attack, even of the epileptoid stage, it may be instantly arrested by ovarian compression. (5) These attacks are not influenced by such treatment as does good in epilepsy. If they are cured, it is by the treatment suitable for hysteria.

The crucial test of Charcot, compression of the ovaries, is rarely successful in this country. An attack can scarcely ever be thus induced, and although it may sometimes be arrested by this means, the effect is not sufficiently constant to possess any diagnostic value. The effect of ovarian compression is considered in greater detail in the next section.

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[1] *Études cliniques sur l'Hystéro-épilepsie*, Paris, 1881.

### ILLUSTRATIONS OF HYSTEROID FITS.

Some of the characters of the attacks, of which a general description has been given, may now be considered in greater detail, and at the same time their features, as they occur in this country, may be illustrated by the narration of some cases. Most of these illustrate many points, so that it is impossible to arrange them in a strictly systematic order.

*Post-epileptic Hysteria.*—Instances of the occurrence of hysteroid convulsion as a sequel to true epileptic fits may be first mentioned.

A pale delicate-looking girl, aged 17, whose father had been insane, had suffered from fits since six years of age, at intervals of two to eight weeks. All were severe attacks; without warning, she fell with a scream, was convulsed, bit her tongue, and slept afterwards. About the age of puberty the fits, as they occurred before, became succeeded by hysteroid convulsion. In one which was seen, she suddenly fell with a scream, the head was turned to the left, the limbs rigid, the face became cyanotic, clonic spasm succeeded, she frothed at the mouth, and bit her tongue. The fit lasted exactly two minutes and a half. She then lay unconscious, breathing heavily, and still frothing at the mouth, for three minutes. Then she opened her eyes, looked strange, kicked and threw her arms about, and dashed her head down and arched her back for four minutes. She then seemed more conscious, but presently again went to sleep, and slept heavily for an hour, and then woke up with a severe headache.

A girl aged 14, without inherited tendency, became, at 13, subject to fits, evidently, from the description, severe epileptic fits, accompanied with tongue-biting. The severe attacks ceased, but attacks of *petit mal* continued, of which she knew nothing, and these, after a time, were succeeded by hysteroid convulsion. She would suddenly bend forwards, unconscious, be laid on the floor and lie still for a few seconds, without any rigidity; then become stiff and begin kicking with her feet and fighting with her hands for a few minutes, and then could be roused. At other times she often had the globus hystericus.

A girl, aged 14, who formerly had severe epileptic fits attended by tongue-biting, came under treatment for seizures of the following character. While speaking to me she suddenly stopped, and bent forwards, remaining still for a few moments without rigidity—a very common form of *petit mal.* Then she suddenly began fighting with her arms and stamping with her feet, and became stiff and rigid. The attack lasted only a few minutes, and she afterwards remembered nothing of what had happened. At other times she also suffered from the globus hystericus.

Hysteroid convulsions often come on during sleep. In some of these cases there is reason to believe that the attack commences with an epileptoid seizure. In others there is no evidence of this. The occurrence of these attacks in sleep need excite no surprise, when it is remembered that the state of brain during sleep is probably not far removed from its condition after an epileptic seizure, and that emotional disturbance, to which hysteroid attacks are often due, is frequently intense during the condition of dreaming.

*Modes of Onset.*—'Warnings' are common before attacks of hysteroid convulsion. In many cases with a distinct aura, it is probable that the aura is really that of a true epileptic seizure, to which the hysteroid convulsion is a sequel. But an aura may occur before hysteroid attacks in which no epileptic element can be discerned. The most common warnings in such cases are a general feeling of malaise and illness, an epigastric sensation, palpitation of the heart, giddiness, some cephalic sensation, an aura referred to both feet, and especially a sense of constriction in the throat, allied to or identical with the 'globus hystericus'—a sensation which is not however confined to these cases (see *antea*, p. 57).

Patients who are aware of the onset of an attack are often able to ward off its occurrence, sometimes altogether, sometimes for a time, by exertion, excitement, or by an effort of the will. I shall presently mention the case of a little girl who, when she felt symptoms of a fit, ran to the nurse, and the attack never came on until she reache her protector. The same fact is illustrated by the following case.

A young man, aged 19, who had had many hysteroid attacks bo day and night, preceded by giddiness, one day, when going home from the City to Norbiton, where he lived, felt suddenly giddy while walking to the station (at 5.30), and the giddiness continued for a quarter an hour. He reached the station and got into the train, and then felt 'kind of stupor,' which was so bad that at Clapham Junction he got o and sat there for an hour and a half. He then felt better, got in another train, and went home. As soon as he reached his house (at 7 he lay down on the bed, and the attack at once came on. He made moaning noise, and began talking with great volubility about busines matters; then he became violent and tried to hit his head, bit his lip plunged and kicked for three hours and a half, and then went into sound sleep.

It is probable that in this case the hysteroid convulsion was post-epileptic, the vertigo indicating a genuine epileptic seizure. If so, the case still affords a remarkable illustration of the ability to restrain for a time the hysteroid convulsion.

In most cases there is no change in the colour of the face when the attack comes on. Sometimes there is initial pallor, and occasionally the patient becomes strikingly pale for some minutes before the onset. The fall, as already stated, may be sudden, and is occasionally violent but more often gradual. An initial scream is not rare and is often repeated during the course of the fit. The rigid spasm may not commence until the patient has fallen especially when the fall is gradual. In some cases the tonic spasm distinctly commences before the fall, which is in these cases, more violent, although the attack is entirely hysteroid. When a patient falls violently, unconscious and pale, and remains for a few moments still, with relaxed limbs, before the distinctive hysteroid symptoms come on it is probable that the initial phenomena are those of an actual attack of epileptic *petit mal*, and that the hysteroid convulsion is thus post-epileptic.

*Tonic Spasm.*—When there is a stage of tonic spasm the limbs are usually rigid in extension, the toes pointed downwards. The arms may lie by the side of the body, or at right angles to it, the patient, in the latter case, lying on the back in the attitude termed by Charcot 'crucifiement.' In some cases the arms may be flexed at the elbows. The fingers are usually flexed at all joints, the fists being clenched. The wrists are sometimes also flexed. There is never the 'interosseal position,' flexion at the metacarpo-phalangeal joints, which is so common in epileptic seizures. The duration of the simple tonic spasm is usually one or two minutes. During its existence the foot-clonus can, as Charcot has pointed out, be often obtained by pressure on the sole of the foot.

*Opisthotonos.*—The opisthotonic spasm is one of the most characteristic features of hysteroid convulsion. It is termed the '*arc en cercle*' by the French writers. It rarely occurs at the commencement of the fit. The initial tonic spasm may pass into it, or it may succeed the stage of clonic spasm, and often recurs from time to time during the progress of the fit. It may amount to simple slight arching of the spine, as the patient lies on the back, or it may be such that the patient rests on the back of the head and the heels, and in extreme cases the trunk may then be pushed up by the feet, so that the neck is flexed to the extent that the vertex or even the forehead is the anterior point of support, and it seems almost as if the neck would be broken. Whilst it lasts the patient may suddenly sit up in bed, and then bound backwards into the rigid arch, and sometimes repeats the action with great force many times.

The opisthotonos may occur as the patient lies on the side. The knees are suddenly flexed, the spine bent, and neck bent backwards, in what Richer terms the *arc en cercle latéral.* Of both these forms he has given some striking illustrations.

Many of the cases here narrated illustrate this form of spasm, since it is present in almost every hysteroid attack. It was a very conspicuous feature in the following cases:—

A girl, aged 15, had had frequent attacks of the following characte She suddenly screamed out and fell backwards, without pallor of the fac She lay quiet for a few seconds, apparently unconscious. Then sh moved her hands like a child trying to box, gently at first, and the more violently, in 'fighting' movements, at the same time kicking ou with her legs. The neck and back were at first rigid and straight, bu presently the movements ceased and the back was arched, the patie resting on the head and heels, and from time to time the trunk w pushed up with the feet, so that the neck was bent backwards to such degree that it seemed as if it would be broken. From time to ti she would bound forwards, almost off the bed, and then back ag in opisthotonos. After about five or ten minutes of this she sudde became quiet, but talked strangely, and once alluded to the fit occurring in another person, and said to the nurse, 'Why don't y hold her down?' After a few minutes' quietude she again pas into a paroxysm of spasm, beginning with the shaking of the han This alternation recurred many times. In some paroxysms she tried bite anyone near her, or her pillow. From time to time there w strong internal strabismus, lasting a few moments, and then ceasin The pupils varied, being sometimes widely dilated, sometimes extreme contracted. Such attacks often lasted for an hour or two. In som the paroxysms of spasm were much shorter than in others, lasting onl half a minute. After an attack she had no recollection of it. Thi patient's attacks had always been of this character. She never had any distinct epileptoid seizures, major or minor.

A girl, aged 25, had frequent attacks during the six months preceding her admission. She had also persistent internal strabismus. The following is an example of the attacks. While lying in bed the internal strabismus became more intense, the angles of the mouth were drawn down. Then she suddenly struck out with her arms, while her face assumed an expression of alarm and determination. She became rigid, and turned over on to the left side, and her back and neck became strongly arched. The fingers were flexed, sometimes both wrists also flexed, at other times one wrist extended. This rigid spasm alternated with violent 'fighting' movements. In some attacks the legs were throughout rigid and extended, in others they were, from time to time, kicked violently. The attacks lasted about ten minutes, and then ceased suddenly. There was no talking or biting or evidence of consciousness. Her sight was dim in the intervals (perhaps from spasm of accommodation), but the amblyopia ceased suddenly after a fit. In this case also there was no evidence of any epileptoid seizure.

*Clonic Spasm.*—The movements in the clonic spasm are quick, small in range, maintaining the same frequency, and would often be more accurately termed tremor, or quivering, than clonic spasm, as in the following case:—

A girl, aged 18, without neurotic heredity, had suffered for eighteen months from fits of the following character. While sitting before me the fingers gradually became flexed, the elbows bent, and the hands were jerked very quickly. The legs hung straight out and rigid, the feet extended, the toes pointed downwards. In a little time the legs were also jerked in fine quick tremor-like spasm. Then the spasm ceased; she sat up, looked wildly about, and presently passed into another paroxysm of rigidity and tremor. Strong ovarian compression seemed to lessen it a little, but did not arrest it, although the attacks could be at once cut short by faradisation of the skin.

Sometimes the clonic spasm affects chiefly the orbicularis palpebrarum. It may amount to merely quivering of the eyelids, or to quick opening and shutting movements, occasionally combined with opening and shutting movements of the hands. Both of these are very common and very characteristic of the hysteroid state. They were well illustrated by the following cases:—

A girl, aged 15, had for twelve months one or two attacks a week, of the following character. She fell back rather gently, without any pallor of face, but with internal strabismus, and rigidity of all the limbs in extension. From the first there was rapid opening and closing of the eyelids. Then the arms were thrown about, and she tried to bite those near her. Breathing was noisy, and the face became red. After a few minutes she became quiet and drowsy, but could be roused, and then passed into the same rigidity as at first, followed by similar movements, again to cease and to recur. Each attack lasted for an hour or more, in spite of douching and galvanism, until they were cut short by apomorphia in the method which is described in the chapter on 'Treatment.'

A girl, aged 18, without inherited tendency, came under treatment for attacks which had commenced five months previously. Without warning she would fall back on the floor, being pale at first, and afterwards red. Then, lying on her back, the arms were stretched out at right angles to the body, in the attitude of *crucifiement,* the legs crossed at the ankles, the limbs being rigid, and the eyelids closed. On opening the lids, the eyes were directed straight forwards and the pupils were large, but instantly the eyeballs were turned upwards, strongly converging, and the pupils, with the convergence, became contracted. Then the legs were jerked with great rapidity, the feet being struck against the floor. The movement was small in range. Presently the right arm was moved in a similar manner, and then the left. Then violent opisthotonos occurred, the patient resting on the back of the head and the heels. After a few moments this ceased, the trunk falling suddenly to the floor, and the quivering of the limbs returned, and the eyelids were similarly affected. These phenomena were repeated several times, the face being flushed. Strong ovarian pressure, at any time during an attack, cause the quick clonic spasm to cease, and induced straining of the muscles o respiration, and opisthotonos, which passed off as soon as the pressur was discontinued. No evidence of consciousness could be elicited. After these symptoms had continued for ten minutes, they ceased suddenly. The patient seemed conscious and sat up on the floor, but after a few minutes she fell back again in the same convulsive movements, but they were slighter, lasted a shorter time, and then ceased and did not again recur. She had such attacks every few days. They could be at any time instantly arrested by strong faradisation of the skin, the current being passed from the neck to the hand, and after a few attacks had been cut short in this manner they ceased to recur, and when she left the hospital she had not had an attack of any kind for three weeks.

In the last case, during the first few moments of the attack the state of the patient was very similar to that of an initial attack of epilepsy; but the effect of treatment is opposed to the idea that there was any true epileptic element, and shows that the attack was really hysteroid throughout.

In hysteroid attacks, although patients so often try to bite other people, and may even bite their own fingers, they do not bite the tongue. Tongue-biting is essentially an epileptic symptom.[1] But although the tongue is not, the lips are sometimes bitten. I have known instances in which patients, throughout every attack, persistently gnawed their lips.

The *coordinated movements*, which constitute so large a part of the phenomena of these attacks, are for the most part, as already stated, wild irregular movements, without sequence or serial order. Sometimes, however, some of the movements are repeated in rhythmical manner. It has been mentioned that some of the spasm in the arms or legs may have this character, being comparatively slow in time and considerable in extent. Thus, the hands or feet may beat the floor. The rapid bounding movements of flexion and extension of the trunk, in which the patient alternately sits up and bounds back in opisthotonos (sometimes seen in this country, but more common in France), is an extreme example of this form. Much more frequent are rhythmical lateral movements of the head from side to side.

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[1] Charcot states that the tongue may be bitten during the initial 'epileptic' (pseudo-epileptic) stage of the severe attacks of hysteria major. In this country the statement certainly does not hold good.

Sometimes there are regular flexion and extension movements of the limbs. When the legs are thus affected, the effect is peculiar. They are alternately drawn up and pushed down, so that the patient, if on the floor, may be propelled, head foremost. The movement was peculiarly uniform in several cases, all females, in which I have observed it. An identical form of rhythmical spasm in the legs has been observed in the convulsion of hydrophobia. In one case it was associated with quicker flexion and extension movements in the arms. Much more commonly there is an analogous movement in the arms, the legs being still, and often rigidly fixed in extension. In one patient, after general tremor for fifteen or twenty seconds, the fingers were flexed, then the wrists, and then the elbows; then the arms, thus flexed, were drawn back at the shoulder-joint and thrown out in front, again to be flexed and drawn back and thrown forwards. This movement constituted all the observable motion in the fit.

This peculiar rhythmical movement in the legs was a feature of the hysteroid attacks in one patient, whose case, although published elsewhere,[1] presents so many instructive points as to deserve narration at length. Both epileptic and hysteroid attacks occurred; the organic heart disease was present; the disease succeeded an attack of chorea; and the patient presented a strong inherited tendency to neurotic disease. It is probable that the hysteroid convulsion really succeeded a slight epileptoid seizure.

Kate B., aged 30, single, was admitted in January 1872, on account of convulsive seizures. A brother had died, aged 40, with what was probably general paralysis of the insane. A sister had an attack of chorea, and died four or five weeks afterwards from heart disease. No relations had suffered from rheumatic fever. The patient herself had never suffered from rheumatic or scarlet fever. At thirteen years of age, she had a severe attack of chorea, which came on gradually without assignable cause, and lasted nine months. She believed that it affected both sides equally. Six months after the chorea, she had a fit; a second fit, three months after the first; and they had recurred at intervals of two or three months. The first and succeeding attacks were described by her as being of the same character. Each began with a sensation in the great toe of the left foot; this did not pass to the other toes, but ascended up the foot and leg to the trunk; as it was passing up the trunk, it commenced in the left forefinger. It did not go to the other fingers, but passed up the arm, and thence to the left side of the tongue, in which she had a sensation of tingling. The sensation was accompanied by some movement of the part: a slow 'drawing up.' She then lost consciousness, and could give no account of the subsequent phenomena. Her tongue, however, was often bitten, so it may be assumed there was severe clonic spasm. Sometimes she had a similar sensation in the right hand, passing up the arm to the side of the tongue, but never to the leg; and this sensation was never followed by loss of consciousness or any further convulsive manifestation. When she had a fit commencing in the left foot, the sensation never passed to the right side. Whilst in the hospital, she had several epileptiform fits, of which no detailed account could be obtained, and also several peculiar attacks of coordinated spasm, one or two of which I witnessed. They began with the same sensation as above described, and, when consciousness was lost, the eyes were turned up and to the left; the arms were not affected, but the legs were rigid and drawn up, and she tended to turn on to her back. Subsequently the legs were drawn up and then pushed down regularly, so that, if she were on the floor, she would work, head foremost, from one side of the room to the other. The sensation preceding these attacks was slighter than that which preceded the severe seizures, and was accompanied by an unpleasant feeling at the back of the head and at the heart. The heart's apex was outside the left nipple, the impulse heaving and diffused, and at the apex was a loud rough, rather long, presystolic murmur, terminated by the first sound. The maximum point was the nipple-line; it was conducted in and out; heard on the mid-axilla, but not at the angle of the scapula, where the heart-sounds were clear. The second sound was reduplicated; a brief interval occurred after the second part of the second sound, and before the presystolic murmur. The fits continued in spite of treatment, although they became slighter.

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[1] 'On the Clinical History of Chorea,' *Brit. Med. Journal*, March 30, 1878.

Nine months later, while taking ten minims of tincture of digitalis three times a day, her pulse sank to 48, and some mental symptoms came on. On omitting the digitalis, the pulse rose in twenty-four hours to 72, but the mental symptoms continued. She fancied persons were in the room speaking to her, and she answered them, and asserted that she was the Holy Ghost; and under that delusion manifested some tendency to violence, so that it was necessary to send her to an asylum. She recovered after some months, and has since continued in nearly the same state, with occasional fits and the same evidence of organic heart disease. The presystolic murmur, however, has been variable; sometimes not to be heard, sometimes distinct, as is not uncommon in cases of mitral constriction. There has been at no time albumen in the urine.

A coordinated movement sometimes seen in these convulsions is an attempt to 'dig the hands into the face or throat,' which may result in serious scratches. It is more common in post-epileptic states than in simple hysteroid convulsions. For instance, a woman, aged 36, whose mother was epileptic, and mother's sister insane, had occasional fits since dentition convulsions. They usually occurred in sleep, but sometimes when awake, and then were preceded by a sensation of 'air rising from the pit of the stomach to the head.' In some of them she bit her tongue. In others, however, she merely fell and immediately began moaning, put both hands to her face and scratched it, and pressed her hands against her nose and mouth, as if trying to suffocate herself.

*Laryngeal Spasm.*—A somewhat rare variety of attack is characterised by extreme difficulty of breathing, not the rapid breathing sometimes observed in hysterical patients, but intense respiratory spasm. They are the 'choking fits' of adults, and are termed 'hysterical strangulation' by French writers. It is no doubt an extreme degree of the disturbance which, in slighter measure, causes the globus hystericus. In some of these cases there is true laryngeal spasm. The patient with infantile hemiplegia and hysteria, mentioned on p. 136, had occasionally severe attacks, very closely resembling prolonged paroxysms of laryngismus stridulus. One day she came into the out-patients' room in a state of hysterical rapid breathing—80 per minute. In order to ascertain whether it would persist during sleep, I hypnotised her by making her look at the hole in an ophthalmoscope. She was quickly asleep, and then breathed only twenty-five times per minute, but presently woke up in the most intense laryngeal spasm, with loud crowing inspiration, and congested and even cyanotic face. It lasted several minutes, and she occasionally hawked up bloody mucus. It would have been alarming had not its hysterical nature been recognised.

The following are other examples of this form of fit:—

A girl, aged 18, had attacks, the character of which was as follows. She was unconscious of any warning, but an observer could note a slightly frightened expression and restless movement of the eyes just before an attack; then she would suddenly fall, and instantly begin labouring for breath. If placed on a chair, she could sit up, but the agony of dyspnœa was extreme. Her head was thrown back, her mouth half open, her arms held out before her with fingers separated; the muscles of inspiration were thrown into violent action, and a choking noise in the throat accompanied each laboured breath. From time to time there was an ineffectual attempt to swallow, and she would strike the upper part of the chest with violence, as if trying to shake something down. Her face rapidly became flushed, then dusky, and the dyspnœa increased, until she seemed on the point of suffocation. By closing the mouth and nose, and so producing actual apnœa, the spasm could generally be stopped, but her aspect was wild and she spoke strangely, and in a few minutes passed into another similar paroxysm. Even if placed under the influence of chloroform the dyspnœa returned as the effect of the anæsthetic passed away, and, if not stopped by means to be described, such attacks would go on for hours.

A girl, from 17 to 18 years of age, suffered from attacks which came on during sleep. Her mother, sleeping with her, would be waked up by a 'suppressed choking scream,' and find the girl unable to get her breath. She became dusky, then very pale, and then the spasm passed away.

It is instructive to compare the features of such an attack with the other well-known forms of respiratory spasm—the globus hystericus, the epigastric-throat aura of epilepsy, the paroxysms of laryngismus stridulus, and the respiratory spasm which constitutes so prominent a feature in the symptoms of hydrophobia.

*Pharyngeal spasm* also occasionally occurs, and may render deglutition impossible. It has been described, in France, as the 'hydrophobic form of hysteria.' The patients may be tormented with thirst, and yet unable to swallow a drop of water. I have only met with slight examples of this variety, but a graphic description has been lately published by Raynaud[1] of a case in which laryngeal and pharyngeal spasm coexisted with trismus, and the patient died in one of the terrible paroxysms of dyspnœa. The case is not altogether beyond criticism, but the balance of evidence is in favour of the accuracy of the diagnosis; the patient presented various hysterical manifestations, and a perfectly similar attack had occurred previously and passed away. The patient had, in the interval, become addicted to the hypodermic injection of morphia, and Raynaud suggests that it may be to the effect of this on the nerve-centres that the fatal termination of the attack was due.

*Eyes.*—The eyelids, in hysteroid seizures, are sometimes separate, more often closed firmly. The eyeballs, as already stated, frequently converge strongly, as in several of the cases related, or are rolled up or to one side. The pupils vary, often they are large; sometimes they vary in size at different periods of the attack. They usually contract during the convergence of the eyeballs, and perhaps without convergence, from spasm of accommodation. The *reflex action* to light, when it can be tested, is usually preserved; but it is most difficult to make observations on this point, since the eyeballs are usually rolled up, and action to light cannot be satisfactorily tested unless the light falls on the macula lutea. Reflex action from the conjunctiva is usually lost or greatly diminished during the severer period of the attack. At other times it is normal. The loss seems to be part of the general diminished sensibility of the surface. In most hysteroid attacks a pin may be run into the skin without any expression of pain, and the conjunctiva may be touched without any contraction of the orbicularis. Sometimes there is a slight muscular contraction when the cornea is touched, but none when the finger is placed on the conjunctiva over the sclerotic, showing that sensibility, although lessened, is not abolished. So, too, a though the skin may be insensitive to the prick of a pi strong faradisation may be acutely felt. In the followi case a careful observation on the pupils was made :—

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[1] *L'Union Médicale*, March 15, 1881, p. 433. The discussion on the case at the Société Médicale des Hôpitaux will be found in the same number.

A girl, aged 25, was admitted, after having been in almost continuo fits for a fortnight. She had no attacks for about three weeks after h admission, and then seizures occurred of the following character. S would fall gently, and lie flat on her back with the eyes open, the ja separated and rigid, the arms by the side, and the arms and legs s Then the arms, still rigid, were raised and dashed down, and the l slowly drawn up and suddenly pushed down (as described on p 15 The movements occurred at regular intervals of about half a mini The attack was readily arrested by closing her mouth and nose. She l also attacks without this rhythmical movement, and one of these witnessed by the resident medical officer (Mr. Frankish). She i apparently unconscious, with body and limbs perfectly rigid, and mo firmly closed. The eyelids were widely separated, and there was oc sional twitching of the orbiculares palpebrarum and occipito-fronta The eyeballs were slightly turned upwards. The pupils were eq dilated, 6 mm. in diameter. They reacted to light, although the c junctiva was insensitive to touch. The respiration was quick shallow. A little later the reflex action from the conjunctiva had turned. The attack was not cut short, and lasted three-quarters of hour, after which she slept soundly for half an hour.

Hysteroid attacks are, as several of the cases alrea mentioned show, common in young girls at ten or twe years of age, and some of the cases which occur at t period are of great severity. One of the most severe ca I have met with, in which the manifestations of hyste were not only intense, but varied and prolonged, occurr in a girl of ten, and the case illustrates so many poi in the symptomatology of these cases as to deserve scription at some length. Especially important is the p longed and peculiar mental disturbance, a feature wh will be again alluded to.

The child belonged to a talented but highly nervous family, altho there was little indication of pronounced disease. She had a sympath mother, who was convinced that her daughter was suffering fro tumour of the brain, and would never give up the charge of her to other person. The illness had commenced six months before I saw soon after a severe mental shock (the death of her father). She ha first attacks of intense 'shaking in the limbs,' succeeded by violent delirium, and she screamed with pain in her head. After this there was persistent mental disturbance (to quote a note of Mr. Harrison Law, who then had charge of her), 'rambling in talk, associating surrounding objects with incidents which she had been reading, and fancying every one would harm her. From time to time she had attacks of rapid flexion and extension of the legs, throwing about of the arms, and catching at the bed-clothes. On some days she would speak to no one.' The mental disturbance improved, but the attacks continued for about a month, and she complained of pain in the left parietal region, where there was at one time intense superficial hyperæsthesia. Soon after this she had attacks in which she would lie quiet for a few minutes, and then cry violently. Sometimes, in the intervals, she did not recognise her mother and sister, but spoke of them as other persons, and occasionally, after speaking strangely, she would scream as if in the most frightful agony, and then suddenly become rational and say 'I think I have been asleep.' About six weeks after the onset she seemed for some weeks in a state of depression. Subsequently her mental condition became quite natural, and the attacks ceased, but she complained that she could not see with the left eye.

When I saw her on June 9, 1879, four months after the onset, she looked and seemed perfectly well, mentally and bodily, with the exception of the loss of sight of the left eye. Vision was R $\frac{1}{1}$, field normal; L $\frac{1}{20}$, with concentric limitation of the field, objects being only recognised just around the fixing point. Colour vision unfortunately was not tested. Ophthalmoscopic appearances normal. The other special senses were normal, except that hearing was not quite so acute on the left side as on the right. There was no hemianæsthesia. A few days later she complained of frontal headache, and one evening was found turning over the leaves of a book, and she took no notice when spoken to, and even when shaken or douched with cold water. In the course of an hour she spoke, and then seemed better, but a little 'queer.' A few days later, although usually a very affectionate child, she became spiteful, passionate, and mischievous. When I saw her a day or two afterwards, she was listless, but the sight of the left eye had become perfectly normal; she could read $\frac{1}{1}$, the field was normal, and colour vision was perfect.

The account of her subsequent symptoms is chiefly from excellent notes which Dr. R. W. Wilson, then of Surbiton, under whose care she had passed, kindly kept for me. Two days later he found her in an apathetic state, not speaking, but slowly eating and drinking what was given to her. In the evening there was a recurrence of fits, consisting of the following series of events:—(1) The eyelids were widely separated, the pupils being dilated and sensitive to light. (2) The eyes were gradually closed. (3) The head was rotated from side to side on the pillow, the movement to the left being always the more forcible. (4) The left forearm was alternately flexed and extended on the arm, the fingers of the hand being extended, and the left leg drawn up and kicked down, the toes being kept extended. Each of the above stages lasted only three or four seconds. (5) Sudden relaxation and immobility for ten or fifteen seconds, broken by a few moans during respiration. (6) She turned on to the right side, and the fingers and toes became strongly extended and drawn back. This tonic spasm gradually passed on to the wrists and forearms, when suddenly violent clonic spasm occurred in all parts of the body except the face and the hands, which continued extended and stiff. There was no cry, or tongue-biting, or foaming at the mouth. After a few seconds there was sudden cessation of the spasm and general muscular relaxation for a few minutes, and then followed another attack with just the same series of phenomena as above described, the only difference being that occasionally the right leg was affected in the first stage as well as the left. During the intermissions her face was flushed and her feet cold, and she occasionally repeated in a very low voice: 'Four cost a penny.' On enquiry it was found that the only time she had spoken during the day was to ask what some sweetmeats had cost, and that was the answer. She seemed to be everywhere insensible to the prick of a needle, and pressure on the ovaries had no effect. After several of the attacks had occurred, Dr. Wilson passed a linen band around the arm near the axilla, with a knot over the nerve trunks, and as soon as the head began to roll, the band was pulled tight, and the attack was immediately arrested, and no other attack occurred that night. She slept well, but the attacks came on in the morning before she was thoroughly awake, and forty occurred before 11 A.M., all being the same as those described. Again they were arrested by the ligature and ceased. During the day she was more herself, but rather deaf. In the evening, she repeated some poems, as Wordsworth's 'We are seven,' and did sums aloud. This went on until midnight, when she had a few fits and went to sleep. A few more occurred before waking in the morning, and then she seemed quite well, and remembered nothing of what had occurred on the preceding day. Some more fits occurred in the evening, and next morning she had fifty-three between 8 and 11 a.m., when they were again arrested by the ligature, and she became conscious, and remembered nothing of the attacks. They recurred in the evening with more repeating of poetry. She had twenty-two fits in forty minutes. So she went on for several days, when she again lost the sight of the left eye, and became slightly deaf in the left ear, for a few days. She complained still of pain in the left parietal region, relieved by a hot sponge. On several days, generally after an attack of pain in the head, her aspect, tone of voice, and manner became quite idiotic, but the mental condition generally passed away with sleep. She had one or two attacks of epistaxis. After several days of freedom from fits she had some more of a somewhat different character, the limbs, including the hands, being all strongly flexed, the eyelids open, the eyeballs strongly converging, and the mouth drawn towards the left ear, all in tonic spasm, but the head was constantly moved backwards and forwards. After a few seconds of this, there was sudden relaxation for a time, and then restless rolling about in bed and muttering, until another attack of the tonic spasm. These attacks recurred for several days. She had much pain in the head, and some ulceration in the mouth. She then had some opisthotonic fits, which commenced by fixation of the jaws. The condition continued on and off for about a fortnight more, and then, about six weeks after the onset, she began to gain strength, the attacks became slighter and briefer, and in the daytime she seemed perfectly well. After a month's freedom from attacks they recommenced, being of the same character, and accompanied by the same idiotic state. This outbreak lasted only a fortnight, and she then became apparently well, and continued so until November. She then had a series of violent convulsive attacks, of much the same character. An initial period of rigid spasm, with the arms sometimes up over the head, sometimes by the side, was sometimes succeeded by pseudo-epileptic clonic spasm, and always followed by violent coordinated movements, in which she rolled from one side of the bed to the other, and, on meeting with an obstruction, rolled back again. When not prevented she rolled out of bed on to the floor. In these attacks the ligature only kept quiet the arm to which it was applied. This series lasted only a few weeks, and since then, with the exception of a few similar attacks while cutting a tooth, she has remained, now for three years, perfectly well, mentally and bodily.

*Ovarian compression*, which is so effective in inducing and in cutting short the attacks of hystero-epilepsy at the Salpêtrière, often, as already stated, fails to produce a marked effect in patients in this country, although ovarian tenderness is by no means uncommon. In such patients, evident distress, choking sensations, and even the feeling by which attacks are heralded, may be produced by compression of the tender ovary, but I have never known such pressure to produce an actual attack. Several instances of this have been already described, and I may mention one or two others. In a girl of sixteen, with well-marked hysteroid attacks, firm ovarian compression caused pain in the head, faintness, and drooping of the eyelids, but no semblance of a paroxysm. In another patient, aged 25, in whom there was marked ovarian tenderness, pressure caused the breath to become short, the face to flush, and a peculiar feeling in the chest, such as often preceded an attack, but the compression never actually induced one.

In a case, however, mentioned on p. 149, during the paroxysms of wild movement an attack of opisthotonic rigidity could always be induced by ovarian compression, but not at other times. In the case of hysteroid attacks just described (p. 156, *et seq.*) in which the attacks were arrested by a ligature around the arm, Dr. Wilson on one occasion tried also ovarian compression. At first it had no effect, but after repeating it in several attacks he found that it did arrest them, but even then less readily than compression at the epigastric region. At a subsequent time, however, the ovarian compression had no influence.

*Duration and Termination of Attacks.*—The duration of hysteroid fits, when not arrested artificially, is very variable. Tney may last only a few minutes, but such brief duration is rare except in cases in which the hysteroid attack succeeds a true epileptic seizure. Unless cut short they often last ten, fifteen, or twenty minutes, and occasionally one or two hours.

They may be often arrested artificially by means which will be described more fully in the chapter on 'Treatment,' viz., closure of the mouth and nose, an abundant affusion of cold water, or painful impressions, of which faradisation of the skin is far more effectual, in this country, than ovarian compression. In some very severe cases these measures fail, and in such I have found the induction of nausea, by a hypodermic injection of apomorphia, to be invariably successful.

The post-epileptic hysteroid convulsion can be arrested by these means as readily as the primary hysteroid attack. This sometimes leads to a mistake in diagnosis. It is said that a patient's fits were not epileptic because they could be stopped by water thrown on the face. An attack may thus be stopped, although the case is one of genuine epilepsy, the fact being that it is not the epileptic fit, but the post-epileptic hysteroid stage which is thus cut short.

The spontaneous termination of an attack is usually sudden; sometimes it is accompanied with a deep sigh, more often it is marked by a few minutes' sleep, at the end of which the patient sits up, and by the return of the natural aspect and tone of voice the friends know that there will be no recurrence. Occasionally the transition occurs suddenly without any other symptom.

Some patients seem to be aware of the termination of an attack; indeed, it occasionally seems as if the cessation was due to an effort of the perverted will, as in the following case:—

A girl who had had a slight attack of hemiplegia at 3, became at 7, after a fall, liable to fits, and these continued until she came under treatment at 14. Whatever the earlier attacks had been, those which occurred under observation presented no epileptic character. They began with jerking of the head, and after this had lasted for an hour she fell down, and began kicking and struggling, throwing her head from side to side; occasionally she would take her pillow between her teeth and try to bite it, and try to tear her clothes. From time to time she rose up, and then dashed her head against the ground. Occasionally she laughed, and talked about a quarrel she had had at home. Suddenly she said, 'Now I won't talk any more.' She then slept for a few minutes and was all right, remembering nothing of the attack. She also had attacks of simple general rigidity (apparently hysteroid), and also some seizures in which there were quick flapping movements of the hands.

*Death in Hysteroid Attacks.*—As a rule, to which exceptions are infinitely rare, hysteroid attacks, however severe and alarming in aspect, are devoid of danger. The attacks of laryngeal spasm present the greatest apparent risk to life, and such paroxysms of dyspnœa as were presented by the hemiplegic girl mentioned on p. 153 are really alarming in appearance even to those familiar with them. That they are not absolutely devoid of peril is shown by Raynaud's case, mentioned on p. 155; but this, I believe, is the only recorded instance of death in an attack of this description.

There is, however, another source of danger—the tendency to turn on the face which is sometimes seen in the post-epileptic state. It is, strictly speaking, rather an automatic than a hysteroid phenomenon; but the two conditions merge into one another; it may coexist with distinct hysteroid symptoms, and may occur as part of the pure hysteroid attacks. An example of death from this cause has been already mentioned, and in the following case the same symptom was present. The patient was a little girl aged 12, who had never suffered from epileptoid seizures, and had had an attack of hysterical paraplegia, immediately cured by strong faradisation. Her fits always occurred on waking out of sleep, before she was quite awake. They commenced by a half moaning, half singing noise, and the louder this was, the worse was the subsequent attack. Then she gave a sudden spring and always turned on to her face, and presently began to scratch the pillow. Sometimes she would rise up in bed suddenly, and then dash her head back on to the pillow. The attacks lasted only a few minutes, and always ended suddenly.

I have met, however, with one instance in which death occurred in consequence of attacks which appeared, to those who witnessed them, to be hysteroid, but were perhaps of the intermediate form described on p. 176. The case was that of a little girl aged 8, who had had attacks for three years, which began with a feeling of fright and giddiness, as if her head were being turned to the right. If at a distance from any persons she always ran to them, and the convulsion never commenced until she got to them, however far she had to run. Then there was champing of the jaws, and the eyes were turned up so that the cornea was invisible, hands and elbows flexed, and the arms moved forwards and backwards at the shoulder for five or ten seconds. Such attacks sometimes occurred every five minutes. One day (at home), after a hearty breakfast, she had a slight fit, then a more severe one, and then, after some hours, another. All were of the same character as those in the hospital, but accompanied with profuse perspiration. She had had at home many similar series of long duration, some lasting nine hours. This series lasted for about four hours, and at the end of that time a rattle came in her throat, and she died almost immediately. No post-mortem examination could be held. It would perhaps be erroneous to regard this death as the direct result of the convulsions. It was possibly due to some intercurrent accident. The case is, however, here mentioned to show that attacks which are apparently hysteroid seizures cannot be regarded as entirely devoid of incidental danger.

*Temperature.*—Hysteroid attacks, however severe and repeated, as Bourneville has shown, rarely raise the temperature, and never more than a degree or fraction of a degree. In this respect they differ from true epileptic fits, several of which, occurring in rapid succession, may cause a considerable elevation of temperature.

In the patients who suffer from these convulsions, other symptoms of hysteria are frequent, and consist of the globus hystericus, aphonia, and the like, but these are usually slight in degree. Occasionally the patients present hysterical paralyses, contractures, or anæsthesia; but such symptoms are, on the whole, infrequent, and it is certainly more common, in this country, to meet with them apart from, than in association with, chronic convulsions. Of paralyses, paraplegia is perhaps the most frequent. Hemiplegia is rare. Spasmodic closure of the jaws is more common than contractures of the limbs; but instances of both these, in males, will be described in the next section. Hemianæsthesia is far less common than among the cases which occur in France. A well-marked example has been lately under my care in a girl of 18. The anæsthesia affected the special senses as well as the limbs, but the phenomena of '*transfert*' could not be obtained. The hysteroid convulsions were severe and characteristic. There was no ovarian tenderness, and ovarian compression had no effect, but there was extreme deep-seated tenderness just below the spleen, and pressure there induced or arrested attacks.

## CHAPTER VII.

### *HYSTEROID ATTACKS—CONTINUED.*

### HYSTEROID ATTACKS IN MALES.

Convulsive attacks of this class are not very rare in lads and young men. They may succeed epileptic seizures in the manner already described, or may occur independently. The subjects of them may present other symptoms of hysteria, such as transient paralyses, or contractures of a limb, precisely similar to those met with in the female sex. The convulsive attacks present the characters already described, tonic and quick clonic spasm, opisthotonos, co-ordinated movements, and delirium with therio-mimicry. Their severity is often very great; the frenzied violence of the movements and mental disturbance may be extreme, while the sex and strength of the patients render their management a matter of even greater difficulty than in the case of females. The convulsions in the following case were among the most severe of the kind I have ever witnessed:—

A young farmer, 15 years of age, six months before, had a 'fit' in a dark cellar into which he had gone alone. Not returning, search was made, and he was found insensible, with a cut head. Whether he had fallen in a fit, or accidentally, could not be ascertained, but a few hours later a distinct fit occurred, and subsequently others. They were stopped by treatment, but recurred after a fright from a horse running away. They were often produced by excitement or by being startled, and he knew that an attack was coming on by palpitation of the heart. I was standing by him when one commenced. First one hand, and then the other, and then his trunk and legs, became rigid. The mouth was twisted to one side. After a few seconds quick clonic spasm came on, commencing suddenly in all his limbs, and continuing with the same rapidity.

In a few moments it ceased. Then he looked frightened, glanced around suspiciously, but tried to put out his tongue when told. Presently another attack of similar character came on, tonic followed by quick clonic spasm, and then a third, till he had had twelve. During the intervals there were manifestations of intense terror; the lad would rise up in bed, look fearfully to one side, catch hold of the attendant, whom he beseeched 'not to let him be hurt.' When the attack, or series of attacks, was over, he seemed to remember what had been said to him in the intervals, but did not recollect his own terror or the convulsive paroxysms. All his attacks presented the recurring tonic and clonic spasm, but the degree of maniacal disturbance in the intervals varied. In one attack, for instance, after the tonic and clonic convulsion was over —to quote my notes made at the time—'he lies quietly in bed for a moment, then suddenly fixes his eyes on some person near, or on the ceiling, or on some part of the room, and stares intently. A look of horror comes gradually over his face, becoming more and more intense as he slowly raises his head. Every muscle becomes fixed and rigid in an agony of terror; the sterno-mastoids stand out in the neck like bars of iron; his eyes seem as if starting from his head, his nostrils are dilated, and his breathing short and quick, while his heart beats with extreme rapidity. Suddenly he begins to struggle, to kick out with his legs, and to strike with his fists with tremendous force, from time to time throwing his arms and head back, still with the same expression of alarm. In a moment he is again rigid in tonic spasm, followed by the same quick clonic convulsion. When this is over, the face assumes a calmer aspect. Presently he starts up in the same paroxysm of terror, each inspiration now accompanied by a strange, weird cry, and his lower jaw drops from time to time, to be again jerked up. Suddenly he makes a gnash at the attendant's hand; failing to catch it he seizes the corner of the sheet between his teeth, and, throwing his head back, shakes it as a dog would shake a rat.' This attack continued, presenting the same alternation of symptoms, for an hour and a half. On two occasions after an attack the jaws were closed in rigid spasm, so that it was impossible to separate the teeth; and this continued for three or four days until another attack, after which it had disappeared. On one occasion he had paralysis of the left arm for three days, which also passed off suddenly. The attacks ceased under treatment, but recurred after the patient had left the hospital, and continued occasionally, until a violent attack of unilateral lumbar pain, very like that attending the passage of a renal calculus. After this he had no other fits, and was heard of, five years later, perfectly well.

Attacks of a somewhat similar character occurred in a lad aged 15. Three months before coming under treatment (having been previously well) he had a prolonged encounter with some burglars, who broke into a house of which he, with some maid-servants, were left in charge. The lad had kept up for hours his endeavours to prevent the thieves from taking away their booty, and received several blows on the head, which stunned him for a time. He was found by the police next morning in some sort of fit; and fits recurred almost daily of the same character as those which he had after admission to the hospital, and of which the following is my note. 'He falls forward suddenly, turns over on his back, and immediately begins to struggle, throwing his arms about violently, and kicking. The eyes are sometimes open, sometimes shut. The eyeballs converge. From time to time the eyeballs are turned upwards or to one side, his face assumes an expression of terror, and he shouts out "Murder! Robbers! Murder!" and then the struggling recommences. The rapidity and force of the movements of the arms and legs can hardly be described. There is no opisthotonos. His head is usually bent forwards, the jaw champed violently, and he is constantly trying to bite whatever is near him. Sometimes, after trying to bite, he makes a barking noise like a dog. After this has gone on for a quarter or half an hour, he suddenly curls himself up on his side, goes to sleep for a few minutes, and wakes up quite well, remembering nothing of the attack.' In some attacks he made movements with the arms as if swimming, saying at the same time, 'Swim away, swim away.' The fits ceased entirely under treatment, and he remained well for four years, and then they recurred, similar in character but slighter. Treatment in hospital soon arrested them, and up to the present time (four years later) he has remained perfectly well.

The tendency to press the fists against the face, which has been mentioned as occurring in some hysteroid attacks in women, is also occasionally presented by males, as in the case of a young man who, at nineteen years of age, had had frequent fits for a year. 'They begin with a feeling of faintness and heavy noisy breathing; he drops on his knees, clenches his hands, and drives his fists into his face. Then he makes a noise something like the bark of a dog, and begins struggling violently, shouts, kicks, and if placed on a bed rolls off it on to the floor, and, if left alone, would roll from one end of the room to the other, knocking his limbs violently against chairs and tables.' The attack lasted only about a minute. Water at once arrested it. The barking noise made by this patient, and which is not uncommon in hysteroid convulsions, is usually associated with the tendency to bite, although the latter may occur alone. The barking and biting are, it will be remembered, prominent symptoms of the hysteroid state that occurs in hydrophobia. In one of my patients, a lad of 13, the attacks commenced after being bitten and frightened by a dog. In the fits the boy would fall, throw his arms out, be still for a moment, and then pass into violent coordinated movements—kicking, biting, &c. He never bit his tongue. The initial symptom resembled the *petit mal* of epilepsy, but he never had any more distinct epileptic seizures, and the attacks were not benefited by bromide. They ceased as soon as the boy was taken into a hospital, to recur when he returned home. Hence this initial stillness was probably only the pseudo-epileptic stage of the hysteroid fit. After the attacks had lasted for several months, the boy's father, putting together the barking and biting, and the apparent cause of the first fit, came to the conclusion that his son was suffering from hydrophobia. Having applied without success to several hospitals, he went to a police magistrate and complained that he 'had a boy who had been suffering for some months from hydrophobia, and no hospital would take him in!' It was in this lad that the animal tendency to bite was carried to such an extreme that he was once found in the act of 'worrying' a cat.

Contracture of one limb during attacks was present in the following case, which illustrates also the transient paralyses succeeding fits, which are occasionally met with :—

The patient was a lad, aged 12, who had had fits for three months, without known cause. There was no neurotic family tendency. A history was given of some epileptic seizures before admission, but those which occurred in the hospital were purely hysteroid. Several attacks occurred daily, and each lasted a considerable time, an hour or more. They began with violent flexion and extension movements of the hands, followed by paroxysms of violence, in which he would spring up, grind his teeth, try to bite, throw his head and arms about, and then lie quiet, except for frequent laboured respiration, and violent contraction of the abdominal muscles, until he again started up. Throughout most of the attacks the right leg was drawn up, so that the heel was in contact with the middle of the opposite thigh, and if pushed down it quickly drew up again. Transient paralysis occurred after some of the attacks. For instance, one morning at 8 a.m. he had a slight fit, merely opening and shutting the hands, and when it was over the right wrist was dropped and powerless. After a similar attack at 11. the other hand was powerless, and the right foot was also powerless and everted. In the afternoon he had another fit, the paralysis continuing, and at 6 p.m. he had an attack of violent general rigidity, followed by struggling, and when this was over, the paralysis had entirely passed away. It returned, however, after another fit, an hour later. During the night he slept, but if touched would strike out with his arms and kick, and grind his teeth, without awaking. In the morning he had two or three fits, but the paralysis was unaltered until the arms and legs were strongly faradised, with the effect of completely restoring power in them for a short time; but the power gradually lessened, and in an hour the limbs were as weak as before. During the day he had several slight fits, without any alteration in his limbs, but in the evening he had a more severe attack, and after this he was perfectly strong. Some weeks later he had some transient recurrence of the paralysis, but under firm treatment the attacks gradually became slighter and ultimately ceased.

Opisthotonos is less common in the hysteroid attacks in males than in females, but is occasionally met with, as in the following case, which is an illustration of the occurrence of these convulsions in an adult man:—

The patient was a railway porter, 28 years of age. Six months previously a heavy weight fell on his head; he was stunned and insensible for four hours, delirious for two days, and had much pain in the head for three or four days more. Two weeks later he had the first fit. He did not bite his tongue. The fits recurred at intervals of a few days; they were said to be preceded for two or three hours by 'jumping sensations' at the bottom of his back, jerking his head and spine backwards, and sometimes a severe jerk of this kind would throw him down. Occasionally a tingling sensation, beginning in the feet, and passing up the trunk to the neck and hands, preceded a fit for an hour. There was never tongue-biting or micturition in the attacks. The patient's manner was nervous and excitable. He had an attack while Dr. Beevor, the resident medical officer, was taking his history. He suddenly complained of 'hot and cold shivers' running down his back, became confused, but not unconscious, lay down on the sofa, and then his back became arched, the head being thrown back, the limbs stiff, and the hands clenched. He rolled off the sofa on to the floor, and rolled about on the floor from one side of the room to the other, the limbs being thrown about violently, the hands clenched, and the head banged on to the floor. After ten minutes he was brought round by flapping his face with a wet towel. Dr. Beevor could not discover the slightest indication of an initial epileptic attack. He was conscious till he got on to the sofa, but remembered nothing afterwards. He was treated first by injections of sclerotic acid, and became less nervous, but several fits occurred, one being excited by a thunderstorm. He was then treated with tincture of mistletoe, and had, after this was commenced, only one slight fit, and finally left the hospital after having had no attack for two months.

It is very uncommon to meet with characteristic hysteroid convulsions in a man over thirty, but they occurred in one patient, an engineer, at the age of thirty-two:—

The first attack was after the extraction of a tooth, but he was said to have been out of his mind for two or three days previously. The fit was followed for a month by several others daily, of which he could give no account except that he talked in them as if he were swimming in the water. (Compare the case on p. 167.) The attacks ceased for eight months and then recurred, and had continued weekly until he came under treatment a year and a half later. A fit which he had in my presence was of the following character. He fell flat on his back, and instantly, without a moment's interval of quiet, struck his arms and legs and the back of his head violently and repeatedly against the ground. The movements had a purposive, *i.e.* coordinated aspect, and was quite unlike the spasm of an epileptic fit. They were of great violence. After a minute or two, he rather suddenly was better, and sat up, but still looked strange, fixing his eyes in one direction, but presently he was all right. In some other fits, seen by Mr. Broster, the resident medical officer, he was at first rigid, with the jaws firmly set, and making a moaning, squeaking noise for a few moments, with the arms and legs extended, before the violent movements came on. This patient was a timid man, with some difficulty in expressing his ideas, although he was a skilful artisan.

## MENTAL DISTURBANCE IN CONNECTION WITH HYSTEROID ATTACKS.

The mental disturbance which attends hysteroid convulsions is a very characteristic feature of the more severe varieties. It is seen in a very striking form in the French hystero-epileptics, whose convulsions are preceded and followed by hallucinations and emotional delusions, of which graphic descriptions and illustrations are given by Richer. But it is also, in a somewhat different form, a prominent characteristic of many hysteroid fits in this country, as several of the cases, described in the preceding pages, testify. The hysteroid attacks which succeed epileptic fits, and those which occur independently, present little difference in this respect. During a series of fits, in the intervals between the convulsive paroxysms, there is always some mental derangement and often positive delirium. The patients talk in an unnatural manner, may have distinct hallucinations. Events which happen before them are falsely interpreted, and incidents lately past, which have made a strong impression on their mind, are reproduced and dominate their ideas. Their *bizarre* fancies are often more like the inconsistencies of a dream than like the delusions of the insane. In some cases the therio-mimicry is apparently the expression of strange hallucinations to which the tendency to bite may be in part related. But the latter cannot be altogether thus explained, since it is seen in cases in which there is no other evidence of this special form of hallucination. In most cases the disturbance of emotion is very marked. The ecstatic states common in these cases in France are very rare in this country, and erotic manifestations are practically unknown here. The emotion is usually painful—fear, horror, anger; and the convulsive paroxysms appear to be, in some cases, the effect of the culmination of this emotional disturbance.

A close relation exists between these hysteroid fits and epileptic mania. The delirium, which is so characteristic a feature of the attacks, often amounts to actual frenzy, such as is exemplified in several of the cases in males just described. The manifestations of emotion are, in these, more intense, and also more chaotic, than in ordinary delirium. The patient's condition might be described as one of compressed mania, the outbursts of varied feeling and action being too concentrated to permit of deliberate development of mental purpose. This condition exists in primary simple hysteroid seizures, but it also exists in the attacks which succeed epileptic fits, *i.e.* under the same conditions as those in which epileptic mania usually arises.

When a series of convulsions is over, the patient's mind usually returns to its normal state, but sometimes the disturbance persists, in slighter degree, for a considerable time. Occasionally this mental disturbance may come on apart from the paroxysms, as in the case of the little girl mentioned on p. 157. In her, apart from the delusions which concurred with the attacks of convulsion, there were occasional periods of spiteful mischievousness or of a semi-demented state. I have known such patients to have transient periods of suicidal tendency, and even to attempt suicide, with the apparent intention of effecting it. In one case, a boy, aged twelve years, had frequent fits, before and after which he had attempted suicide, having tried to hang himself and to cut his throat. The first fit occurred six months before he came under treatment, on a severe fright. They began with an aura resembling that of epilepsy, a sensation in the left great toe which passed up the body to the head, when he lost consciousness and began 'struggling.' The boy was admitted to the hospital, and iron given, but he never had another fit, and after three months was discharged, and continued well after his discharge.

How profound may be the mental disturbance in some of these patients is shown by the following case, the history of which deserves narration at length, because it illustrates the fact that the mental disturbance of hysteria may pass into a condition of what is practically insanity.

A girl, aged 26, had suffered from fits, evidently, from the description, hysteroid, for six months before she came under treatment. After her admission into the hospital she had several attacks in which she slowly fell backwards, without pallor, and then struggled and threw herself about, being made much more violent by any attempt to restrain her. Water quickly brought her round, and so did strong faradisation. In some attacks she made blowing noises with her mouth, and tore her hair. After a little time the attacks became more severe, and were attended by more mental disturbance, and subsequently paroxysms of mental disturbance came on without any preceding fit. For instance, one day she had a fit, went to sleep for a short time, then seemed pretty well, and got up and dressed herself, and two hours later suddenly began to undress herself, and took off nearly all her clothes. She was prevailed upon to get into bed, but presently got out again, and began to take down the pictures in the ward, and if any persons touched her she attempted to bite, kick, and scratch them. She began to stammer, and in the course of a few days presented muscular weakness, which increased until she was in a most pitiable condition, scarcely able to walk, except during paroxysms of violence. On one occasion she had managed to slip out of the ward unobserved, and was found to have taken off her clothes and to be lying upon the stairs. Under treatment she improved greatly, became strong, and passed a month without an attack. She afterwards had an occasional outburst of such violence, that it was necessary to transfer her to Bethlem.

Dr. Savage has kindly informed me that after her admission into the asylum (February 17, 1877) she remained without an attack, being quiet and industrious, until the morning of April 3, when she suddenly became statue-like, standing fixed and taking no notice of her surroundings. After an hour of this condition she suddenly sprang upon the attendant, got her down, and would have injured her if not prevented. She then passed into a state of hysteroid convulsion, throwing the limbs about wildly, and clutching any persons near her, and trying to bite them. The pupils were contracted, the left more than the right. The conjunctivae were apparently insensible. At 11.30 she was put into a wet pack, and became quiet. The eyelids were then open, and the eyes and head fixed and motionless for several hours, including three-quarters of an hour's ophthalmoscopic examination. She had another convulsive attack at 5.30, and then became quiet and remained so through the night, except that at 5 A.M. she sang hymns for a short time. The next day she was sensible, and remembered nothing of what had happened. Eight days later she had several similar fits, in one of which she bit one of her knuckles slightly. From this time she improved slowly, and at the end of May went to the Convalescent Branch. In June she had to leave, to go to a sister who was very ill, and this upset her for a time. She was readmitted to Bethlem depressed, but began to improve until a fresh shock from the death of her sister. After this menstruation ceased, she became violent, dangerous, and refused food. Under treatment (tepid baths with cold affusion to the head) menstruation returned. Early in 1878 her condition was very unfavourable; she was weaker, and her habits were more degraded, so that on several occasions she was reported to have eaten her fæces. In the spring she again improved, went to the country branch, and became strong and hearty, and in July 1878 she was discharged. A few months later she was keeping quite well, and was in permanent employment.

## THE ASSOCIATION OF EPILEPTIC AND HYSTEROID CONVULSIONS.

Whatever be the relation of the processes which underlie the two forms of convulsive attack—a subject which will be again alluded to—it may be well to recapitulate some facts in regard to the association of the two. The first is that the sequence, the occurrence of hysteroid attacks after epileptic seizures, is occasional and, as already intimated, not constant, and is met with under certain conditions. A large number of patients have major or minor fits without any succeeding hysteroid phenomena, although the attacks are apparently similar to those which, in other patients (and even in the same patients at another period), are so succeeded. Further, those who do present the sequence are at the age, and for the most part of the sex, in which hysteria is common. They are especially lads, girls, and young women. The sequence is excessively rare in adult men, and is practically unknown in this country, in either sex, when epilepsy commences after forty. In many of these patients (but not in all) there are other manifestations of hysteria. Hence it seems that a special state of the nervous system is essential for the occurrence of these post-epileptic hysteroid manifestations. There must be some degree of the condition of which simple hysteria is the manifestation. In some of the patients this condition may be so strong as to give rise to other spontaneous hysterical attacks, and to other symptoms of hysteria. In other patients it may be so slight that it never manifests itself except in the special condition which is present immediately after an epileptic fit. Hence we must regard all patients who have true epileptic attacks, major or minor, and present, after some or all of them, hysteroid phenomena, as being the subjects of both epilepsy and hysteria.

The frequency with which this association exists need be no matter of surprise. Hysteria, it must be remembered, is common not only as an isolated, but also as a conjoined, morbid state. It is the expression of an imperfect cerebral nutrition, and is determined in its special form by the functional condition of the individual brain, a condition which depends upon age, sex, and inherited and acquired peculiarities. When these conditions exist, the state may be set up by many causes. It may be excited by organic disease. Striking symptoms of hysteria are often seen, for instance, in cases of tumour of the brain. A large number of the symptoms of hydrophobia, the form of convulsion, the barking and biting, are essentially the same as those met with in many hysteroid convulsions, while even the respiratory spasm of hydrophobia has its homologue in the throat spasm of simple hysteria. Hence, to use the apt words of Weir Mitchell, the symptoms of many organic diseases of the nervous system are pictures 'painted on an hysterical background.'

It is not therefore surprising that this is true of a disease which has so wide an influence on cerebral nutrition as epilepsy—an influence which is often seen in the extensive degradation of function expressed by impairment of mental power. Hysteria would seem to be, in many cases, the expression of this commencing or slight degradation in a brain which is in the functional state suitable for the development of this disease.

Hence it is intelligible that, at certain ages, not only should epileptic patients present, after their attacks, hysteroid symptoms, but that many of them should suffer from other symptoms of hysteria, and from hysteroid attacks in which no epileptic element can be discerned. The most severe hysteroid convulsions I have seen, attacks which came on without the least epileptoid symptom, occurred at the age of puberty in a girl who had been liable since early life, and continued liable, to severe epileptic fits.

## ATTACKS INTERMEDIATE BETWEEN HYSTERIA AND EPILEPSY.

Many careful observers, as for instance Trousseau, have considered that the characters of some attacks are intermediate between hysteroid and epileptic fits. But the tendency of recent study of these diseases has been to discountenance the idea that there are such attacks, to regard all doubtful cases as belonging entirely to one disease or to the other, or as presenting a serial (and not simultaneous) combination of the two. There is much however in the results of recent investigation to prepare us for the existence of such intermediate seizures. A comparison of the features of many attacks which we regard as epileptic or hysteroid, shows that many deviate very considerably from that which we regard as a typical seizure of either kind. These deviations are for the most part in the direction of the other form of attack. In severe hysteroid fits the initial stage, which, as described on p. 143, so closely resembles an epileptic fit, must be due to a discharge having the same seat as in true epilepsy, although probably differing in its pathological causation. In some epileptic fits, as part of the attack itself and not a sequel, there are phenomena which remind us of hysteroid seizures. I may refer, for instance, to the severe attack described on p. 79. It occurred in a hysterical girl who had also purely hysteroid attacks and others similar to that described. The onset was a sudden violent fall, without any premonitory symptoms; the clonic spasm was shock-like, perfectly epileptic in type, and the aspect of the patient was precisely like that seen in a most severe epileptic fit. And yet there was throughout a tendency to opisthotonos, and very brief hysteroid spasm came on without any interval, as the terminal stage.

Coordination of the muscular contractions which constitute the convulsion is the characteristic feature in hysteroid attacks, the absence of coordination is the characteristic of epileptic fits. But the initial phenomena of some epileptic fits—such, for instance, as the visual and auditory warnings described on pp. 65 and 69—show that the 'discharge' in such attacks, when slowly developed and in certain situations, may be in the form of a coordinated nervous process. The same conclusion is suggested by some other initial symptoms, such as the act of running. An equally complex act preceded the attacks in a patient who had had major and minor attacks since rickets in infancy, and there was a family history of epilepsy. The minor attacks consisted of slight clonic spasm, the major were severe epileptic convulsions attended with tongue-biting. After both he went to sleep, but after the minor attacks, before going to sleep, he always jumped for five minutes, got on a table or chair and jumped off, tried to jump over the banisters. *Both severe and slight fits began with hopping round the room on one leg.*

If the discharge may cause, in its commencement, a coordinated movement, it is not surprising that cases should be occasionally met with in which all the spasm of a slight fit of genuine epilepsy should consist of coordinated movements, *i.e.* that spasm of hysteroid type should constitute the convulsion of a true epileptic fit. I believe that this was the case in the patient whose attacks are described on p. 91. The fits occurred almost daily during several years. Sometimes they were excited by rising from the sitting posture, at other times they occurred apart from movement, both by day and night. There was no warning; the attack was very brief, lasting only a few seconds; the patient was unconscious, the pupils dilated, the conjunctiva insensitive, and after the fit there was almost complete loss of the knee-jerk for a few seconds. But the tonic muscular spasm, always the same, consisted in flexion of the left leg and elevation of the right arm; the right hand made scratching movements, so as sometimes to inflict a number of scratches on the face; the expression throughout was one of extreme alarm, and sometimes the tongue was protruded. Throughout two years the attacks varied very little in character. The character of the spasm, the emotional expression, the occasional protrusion of the tongue, were hysteroid, but the general course of the disease, the sudden brief fits, with complete unconsciousness and dilated pupils, rendered it much more closely allied to epilepsy than to hysteria. Of the latter condition there was no other manifestation. Moreover, the attacks ceased under bromide, and were entirely uninfluenced by any treatment for hysteria. The fatal case described on p. 162 was probably also an example of this intermediate form.[1]

Emprosthotonic spasm, a tendency to 'curl up in a ball,' is a form of coordinated spasm occasionally seen in cases which seem to be of this intermediate class. For instance, a boy aged thirteen had suffered from fits for fifteen months, which commenced with a sudden start of considerable violence, and immediately his legs became strongly flexed, and his trunk bent forwards with the head between the knees. These attacks occurred chiefly in sleep, but he had somewhat similar attacks in the daytime, which commenced by a tingling sensation in the right temple; then he clasped his hands behind his head and bent his head forward to his knees, kicking with his legs the while. He always passed urine during the fit. He also had attacks of sudden faintness during the day, but there was no trace of *petit mal* at the commencement of the coordinated fits, and it was during the progress of these that the micturition occurred. He had twelve or fourteen fits a day on various treatment. It was then found that he practised masturbation; a blister on the prepuce reduced the fits to from two to seven daily. He was then circumcised, and the attacks ceased at once, and did not recur.

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[1] It is probable that these cases are similar to one mentioned by Trousseau, of 'a young girl who during her epileptic fits—which lasted one minute only—was frequently agitated with the same violence and the same sort of jactitation which belong to hysteria.' (Trousseau's *Lectures on Diseases of the Nervous System*, Bazire's translation, p. 91.)

These facts suggest, I think, that the morbid action of the nervous system which causes the visible phenomena of attacks may in some cases present such a combination of the processes which underlie the hysteroid and epileptic forms of convulsion, that attacks occur in which the characters of the two forms are combined at the same time, and are not merely associated in consecutive development. No doubt most of these cases may be placed, approximately, in one or the other group; but they show that the two forms of disease are not separated by any fixed and impassable symptomatic boundary.

There is one other point in connection with this subject to which I would direct attention, but rather as a suggestion for further enquiry than as a definite opinion. The phenomena of some cases of chronic hysteroid attacks seem to suggest that the long-continued recurrence of these seizures may, in certain conditions of the nervous system, lead to such a development of the morbid process that attacks occur which are much more closely allied to epileptic than to hysteroid seizures, and may even be truly epileptic. The 'pseudo-epileptic' stage of hysteroid attacks must, as suggested above, be ascribed to a sudden liberation of energy, a 'discharge' in certain centres, and the degree of impairment of consciousness varies in different attacks and in different stages of the same attack, being in some considerable, if not complete. May not the repetition of this process of discharge so augment the tendency to it, that it occurs after a time with such suddenness and spontaneity, and with such affection of consciousness, that we must regard, as in part epileptic, attacks which were at first purely hysteroid?

Several cases which I have watched suggest this question. As an example may be mentioned that of a spoiled pugnacious boy, with a great dislike to mental application. At the age of twelve he became liable to attacks, the nature of which puzzled his medical attendant. They occurred almost daily. If he had a day's hunting, he was sure to be free. But he never did a day's lessons without one. He always had the attacks when he was with his tutor, never when with his mother, for whom he had considerable regard. He would sit down to work, and after half an hour want to leave off and talk to his tutor. If requested to continue his work, he would presently make some strange, inapposite remark, or, if writing, begin making random marks upon the paper, and then look up with his eyes fixed on some distant part of the room, or get up and walk round the room, with staring eyes, and, if restrained, begin to fight, or abuse those who interfered with him with great volubility. After this mental state had existed for a short time, his eyes would close, and his head sink forward for a moment or two in what resembled an attack of epileptic *petit mal*; then, in a few moments, he would pass into a state of violent hysteroid convulsion, throwing back of the head, kicking and struggling, which lasted for ten or twenty minutes. After he had had such attacks for some time, the different elements occurred sometimes separately; he had occasionally the initial mental aberration without further phenomena, and in it he sometimes threw objects at his brother, which, had they struck, might have gravely injured him. At other times he had the momentary swoons, certainly not simulated, without other symptoms, most closely resembling attacks of minor epilepsy. But they were distinctly induced by mental states. They were not in any degree influenced by bromides; but this is of small value as evidence of their nature, because cases of true *petit mal* are often met with over which bromides exert little influence.

The same question—the possible progress of hysteroid phenomena into disturbance which is more than hysteroid—is suggested by the symptoms of mental derangement in the patient whose case is narrated at p. 172.

# CHAPTER VIII.

## *CERTAIN MORBID ASSOCIATIONS OF EPILEPSY.*

THE several morbid states with which epilepsy may be associated have been for the most part enumerated in the chapter on 'Etiology.' Two of them, however, heart disease and chorea, the relation of which to epilepsy is variable or uncertain, need further consideration.

### HEART DISEASE.

An abnormal condition of the heart was noted in 93 cases, but this does not represent the proportion of the whole in which abnormality existed, since in some its condition was not noted. I have, however, rarely failed to examine the heart in epileptics, and I think that the proportion of cases of valvular disease is not far below the mark. In 8 cases there was extreme frequency of action; in 9 there was irregularity; in 13, all young persons, there was reduplication of the first sound. In 31 cases there was valvular disease, mitral regurgitation in 20, mitral constriction in 7, aortic regurgitation in 3, and aortic constriction in 1. There was obvious dilatation, without valve disease, in 20, and considerable hypertrophy, without renal disease, in 2. The cases of irregularity and reduplication were all observed within a comparatively short space of time, and I am sure that, had my attention been earlier directed to the frequency of these conditions, I should have found them in a larger number of cases. In a few cases it seemed that the heart disease and epilepsy were in accidental association. In some cases the cardiac condition, especially simple dilatation and reduplication of the first sound, may have been a consequence of the repeated strain to which the heart had been subjected during the attacks. But in other cases, especially of valvular disease, there was reason to believe that the heart disease existed before the epilepsy, and in many there was no other etiological condition to which it could be ascribed.

Regarding the nature of the connection between the two, we have no facts to guide us, but it may be explained on more than one hypothesis. Capillary embolism or thrombosis has been suggested as the explanation of the mental disturbance which sometimes occurs in heart disease, but this condition differs from the epilepsy which is met with in these cases in its late occurrence. It usually attends the terminal stages of the cardiac affection when the signs of cardiac failure are pronounced. A slow degradation of cerebral nutrition, in consequence of the imperfect blood supply, is another possible explanation, while there are also facts which suggest that, in some cases, the disease may originate in the medulla, the cardio-vascular centres of which are sometimes manifestly disturbed in their action in cases of organic heart disease. As bearing on this theory, the following case, in which epileptic and anginal seizures alternated, is of great interest:—

Eliza T., aged 28, admitted into the Queen Square Hospital, October 16, 1876. No neurotic family history, or evidence of syphilis. She had had four attacks of rheumatic fever, the last nine months before. Soon after this attack she became liable to severe paroxysms of palpitation, chest-pain, and dyspnœa. These continued for about two months, and then ceased on the occurrence of some convulsive seizures, which recurred, almost daily, up to the time of her admission. She was found to have a presystolic mitral murmur, not always audible. Occasionally a systolic murmur could be heard. The impulse was a little diffused; the apex nearly in the normal situation. The heart's action was frequent, 120–160. The pulse small, and rather hard. There was no goître. The convulsive attacks were said to be sometimes preceded by 'swimming in the head,' sometimes by no warning. In some of them she was said to have bitten her tongue, but those which occurred after her admission into the hospital were not distinctly epileptic in character. The first thing noticed was tremor in the right arm and leg, without change in the colour of the face. After a few minutes the head and eyes were turned to the left, the head was thrown back, the back somewhat arched, and the arms became rigid. After a minute the arms were thrown about, she knocked her head, and pulled her hair. Towards the end of the fit she became a little dusky in the face.

She was at first treated with bromide of ammonium, digitalis, and morphia, and by this the pulse was gradually reduced to 76, but the attacks became more frequent. The digitalis and morphia were then omitted; the pulse steadily increased in frequency, but the fits continued, and by November 5th from three to seven occurred daily. Arsenic was then given instead of bromide, and the attacks were cut short by stopping the breath, and by water. For three or four days two attacks occurred daily, and they then ceased. The pulse, however, continued to rise in frequency, being rarely below 130, and the attacks of dyspnœa and palpitation, which had occurred before the fits commenced, again returned. Many of these were carefully watched by Dr. Allen Sturge, the resident medical officer, and by myself. She would suddenly press her hand over the lower part of the sternum, presently almost stop breathing, with teeth tightly clenched, and an expression of extreme alarm and distress on her face, rocking to and fro, and able to speak only a word or two in a whisper. She described a pain, usually behind the sternum, beginning gradually, and after a few seconds becoming most intense, shooting up to the left shoulder, and sometimes down the left arm to the tips of the fingers. Occasionally the pain was felt elsewhere in the thorax, as under the right breast. Before she was conscious of the impending attack, its approach could be recognised by a change in the character of her pulse. Previously 90–120, moderately soft, it would gradually become smaller and harder and more frequent, and then the pain came on. Towards the height of the seizure the pulse was 190–200, and extremely small and hard, but just before the attack began to subside, the pulse could be noticed to become softer. Nitrite of amyl relieved an attack when flushing was produced, but it was difficult to get this result; bystanders several feet away were flushed long before the patient to whose nostrils and mouth the amyl was applied. The inhalation, however, on several occasions arrested impending attacks. They continued, in spite of treatment, as long as she remained in the hospital. I have lately (1881) learned that the patient is still alive, and in nearly the same condition.

### CHOREA.

Epilepsy and chorea are occasionally associated, and were so in 20 cases of this series. The relation between the two diseases was as follows. In 8 cases epilepsy existed before the chorea. In 12 the chorea occurred first; in 4 of these the fits began at the time of the chorea, and persisted afterwards. In only 2 of these was there heart disease. In 5 cases the fits occurred long after the chorea, the interval being four, six, twenty-seven, thirty, and forty years respectively. It seems probable in the cases in which the fits immediately succeeded the chorea, and possible in the cases in which an interval elapsed, that the impaired nutrition of the motor centres during the chorea may have left a predisposition to further disturbance under the action of some other exciting cause.

The possibility of a close connection between the two forms of spasmodic disorder is shown in the following remarkable case, in which paroxysmal convulsive seizures were part of a severe attack of chorea. In them, although the spasm was in part that of chorea, so intense as to assume a convulsive character, there was also loss of consciousness, and spasm of a tonic character preceding and accompanying the choreoid convulsion. The significance of this case is that the convulsion must be ascribed to the same morbid process as that which was the cause of the chorea.

Alice P., aged 15, was admitted in October 1875, with general chorea, very violent in character. She had had repeated attacks of chorea during the preceding seven years, for which she had been in several hospitals. She had a loud mitral presystolic murmur. At first the choreic movements were greater on the left side than on the right. The attack lasted four months, and then the chorea gradually lessened, almost, but not quite, ceasing for several months, and then increased so that, in October 1876, she was readmitted. There was then much mental obtuseness; dull aspect. When she was at rest in bed, the spasm was slight; on movement, it became extreme and general. The speech was difficult. The tongue was protruded and withdrawn slowly. Almost every day after her admission, she had a peculiar convulsive seizure. One of these I saw, and several were witnessed by Dr. Allen Sturge, then resident medical officer, who made some careful notes of their character. The following is his account of one of these attacks. 'Whilst I was listening to the heart, and just after she had spoken to me, she gave a sort of groan, became flushed and somewhat cyanotic; her head was turned to the right, the left sterno-mastoid being strongly contracted, and the eyes deviated to the right. There was a good deal of general movement of the limbs, at first choreic rather than convulsive. Thinking she was in pain, I asked her what was the matter, but got no answer, and she was apparently unconscious. The movements increased, and she had some degree of opisthotonos for a short time, and the character of the movements became more convulsive. The condition lasted three or four minutes and gradually went off, leaving her in a dull heavy state, and with more choreic movement than before the fit.'

In another fit, which was watched from the commencement, the head again turned to the right. There was no initial pallor of face, but the countenance become more suffused than before the fit. The right arm and leg were affected with greatly increased choreic movements, while the left arm was stretched out rigidly from the side with the fingers extended, the thumb extended and drawn under the fingers. The rigidity was cataleptic in character; for the arm remained rigid wherever it was placed. The fit lasted about two minutes, and left the patient very stupid and dull. After she came to herself, the left arm was weaker than before.

A third fit, witnessed a day or two afterwards, differed in some respects from the others, although of the same general character. She was at first apparently more or less unconscious, without any movement. This condition lasted for half a minute, and then convulsion set in. The head was drawn violently to the right; the eyes remained open, the pupils rather dilated. There was some arching of the back, and tonic spasm in both arms and hands, that in the flexors preponderating, so that the elbow, wrist, and finger-joints were all strongly flexed, and the arm was drawn up almost to a level with the shoulders. After a few seconds the right arm became affected with a sort of choreic movement, the wrist and fingers remaining flexed. The left arm became more flaccid, but remained motionless, while the right arm was affected with this movement. The legs were not specially noticed, but there was certainly not much movement in them. The jaws all the time were firmly closed. The whole condition lasted about two minutes, and she was left dull and stupid. The left arm did not seem weaker after the attack, but a fortnight later it was found that the arm was almost powerless. Both legs were weak, but the left was weaker than the right.

Fits similar to these occurred daily for three weeks. Under hypodermic injection of morphia the fits ceased, and the left arm became stronger, but the choreic movements in it were more considerable. She improved for a time, and then relapsed and remained stationary until glycerine was given (half an ounce three times daily), which was followed by a very rapid and immediate improvement up to a certain point. Afterwards, with country air and zinc, all choreic movements ceased, and she has remained free from the disease, the mitral presystolic murmur persisting. A year later, however, she was under the care of a colleague with another attack.

In the next case, also, the convulsive attacks, distinctly epileptiform, must be ascribed to the morbid process which caused the chorea, since they immediately succeeded the chorea and corresponded to it in distribution.

George C., aged 12, presented himself with a history of a first attack of chorea, which had commenced six weeks before, and had lasted a month, leaving, however, considerable weakness of the legs, that of the right being the greater, and some weakness of the right arm. During the fortnight after the cessation of the chorea, he had had several right-sided convulsive attacks, with loss of consciousness. Under treatment the fits became much less frequent, but did not cease; the muscular weakness, however, disappeared. The patient had no cardiac murmur, but the action of the heart was irregular.

The attacks which succeed chorea are, however, much less frequently epileptic than hysteroid, or of an intermediate form not quite corresponding to either. In the next case, for instance, the first attacks were apparently distinctly epileptic, but subsequent seizures, from their character and long duration, were unlike any ordinary epileptoid seizures.

The patient was a married woman, thirty-two years of age. As far as could be ascertained, there was no family predisposition to nervous disease. She had had good health in early life. At nineteen years of age she had an attack of chorea, occasioned by a fright, and sufficiently severe to cause her admission into a general hospital. The attack was a long one, lasting two years. Two years after the cessation of the chorea, and after some trouble, she had her first convulsive seizure. No fuller account of it could be obtained than that she was stiff, and screamed, and bit her tongue. The fits continued from that time; and she said that of late, when a fit was coming on, objects looked strange to her, and she saw sparks before her eyes, and sometimes an appearance as of smoke or of 'all kinds of colours.' She was admitted into the hospital, and there had attacks in which, with apparent loss of consciousness, there was general tonic rigidity, but no clonic convulsion. Some of the attacks lasted several hours; others were shorter in duration, and seemed to be excited by a severe cough which troubled her.

Another example of the association was presented by the patient whose case is narrated at p. 151. Epileptoid and hysteroid attacks occurred, but the patient suffered also from organic disease of the heart. The attacks in the next case were probably severe hysteroid seizures with an initial well-marked pseudo-epileptic stage.

Ada S., when five years of age, was under my care for a severe attack of chorea, from which she recovered, apparently completely. Three years after the chorea, when nine years of age, she became subject to 'fits.' These attacks commenced with jerking of the head and loss of power of speech for several hours. She was able during this time to hear quite well, but was unable to speak. Then she would suddenly lose consciousness, the jaws 'champed,' a noise was made in the throat, and 'foam' collected in the mouth, almost choking her. There was no general convulsion. After the attack she generally slept for some time. At one time these attacks recurred regularly every three weeks. Under treatment the fits became less frequent and slighter, but there was much jerking of the head after waking in the morning, lasting an hour or more. In the slighter fits she would suddenly fall, be unable to speak for a moment, and rigid; then she would scream, clutch with her hands, and foam at the mouth. There was no cardiac murmur.

In the following cases the chorea succeeded epileptiform attacks, and it is probable that the two were due to a common condition rather than that there was any causal relation between them.

*A Single Epileptic Fit succeeded a month afterwards by Hemichorea.* A lad, aged 17, lately came under treatment for slight but characteristic hemichorea. The affection was of two months' duration, and was first discovered by objects being dropped in consequence of a 'kind of catch in the hand.' There was only a slight occasional twitch in the hand and on the right side of the face, but an attempt at movement was accompanied by characteristic choreic incoordination. The right hand was weaker than the left. (Dynamometer—right 31, left 41 *kilogrammes.*) He had never had rheumatic fever, and there was no cardiac murmur. His mother said that about three or four years ago he had an illness, accompanied by twitchings, and occasioned by fright. Three months before the patient came under observation, and one month before the commencement of the hemichorea, he had an epileptic fit, which seemed, as far as could be ascertained, to have been general. He fell without warning, and during the fit he bit his tongue.

*Left-sided Fits for six months, beginning in Foot: Slight Attack of General Chorea.*—Eliza T., aged 22, had her first fit six months before she came under treatment. The catamenia had never appeared. She had had no food for two days before the first fit. It was the same in character as subsequent attacks. Each began with a sensation in the left foot; the left leg was then drawn up, the knee being flexed, with a series of jerks, and the foot drawn up over the other leg. The sensation then seemed to pass up her side to the top of the head, and then back again to her heart, where it caused a sort of pain, with which she lost consciousness and fell backwards. What happened during the further progress of the fit could not be ascertained; apparently some time elapsed before consciousness was recovered, and on coming to herself she invariably asked the time. Two or three such fits had occurred weekly. For a few weeks before admission, she had noticed a numb feeling in the left arm, and when holding an object the grasp would suddenly relax for a moment, and during about two weeks there had been involuntary movements in the arms. On admission, there were well-marked choreic movements of the hands and head, slight but quite characteristic, spontaneous. There was very little incoordination on voluntary movement. There was no cardiac murmur. After her admission, the chorea gradually ceased in the course of six weeks, and she had no fits, although she sometimes had a sensation, such as preceded a fit, commencing in the foot and passing up to the head. These sensations continued for some weeks after her discharge; but she had no severe fit during three months in which she continued to attend as an out-patient.

*Fits from Infancy, chiefly left-sided: Chorea at Eleven, chiefly affecting Left Side.*— A girl, aged 16, came under treatment for epilepsy, which had existed from infancy. The first attack occurred at six months of age, during sleep. No history of rickets could be elicited. The attacks recurred at first every three months, afterwards more frequently. When eleven years of age, she had an attack of chorea, of three months' duration, succeeding an illness with some symptoms of rheumatic fever. The chorea began in the left side, and afterwards spread to the right, but throughout it was the more severe on the left side. During the chorea she had no fits, but they recommenced immediately after the cessation of the chorea. The fits were preceded by no warning; consciousness was lost. The left hand was clenched and put out, the right being kept still. The mouth was drawn towards the left. There was usually little or no jerking, but sometimes there was a good deal, and it was then confined to the left side. There was no evidence of heart disease.

# CHAPTER IX.

## *COURSE OF EPILEPSY.*

EPILEPSY may commence in one of three ways. First by minor seizures which occur alone for months or years before there are severe attacks. The attacks of *petit mal* are often at first slight and attract little notice, but become more frequent, until suddenly a severe convulsion occurs. The patient and his friends do not associate the two forms of convulsion, and it is always necessary to make careful enquiry for the occurrence of minor seizures, antecedent to that which the patient believes to have been the first epileptic fit. It is common, for instance, for a patient to say that he has had fits for a few months only, when attacks of *petit mal* have been occurring for years.

The second mode of commencement is by severe fits recurring at short intervals, without any preceding *petit mal.* The second fit occurs within a few days or a few weeks after the first, and the attacks recur at short intervals.

The third mode of onset is with a single severe fit, and no other fit or sign of epilepsy for months and even years, when another attack occurs, after which they usually become frequent. Between the last two forms there is every gradation of varying interval between the first and second fit.

*Interval between the First and Second Fits.*—If an individual suddenly has an epileptic convulsion for the first time, for which no cause can be discovered, central or peripheral, it is probably the commencement of epilepsy. Whether it is so or not can only be conclusively decided by the occurrence of another fit. Both patient and doctor anxiously ask: If this is epilepsy, when may another fit be expected? After how long a period of freedom is the expectation justified that no other fit will occur? No exact answer to this question can be given on account of the great variations in the interval between the first and second fit, the modifying effect of treatment in postponing or preventing the second fit, and because we have no means of ascertaining, on any considerable scale, the frequency with which a single epileptic fit occurs without successors. But some help in forming an opinion may be obtained by comparing the relative frequency of interval between the first and second severe fits in a series of cases. Accurate facts on this point could be ascertained in only 160 cases. The result is shown in the following table.

*Interval between the First and Second Severe Fit in* 160 *Cases of Epilepsy.*

| | | |
|---|---|---|
| Less than 1 week | 18 | 55 cases under one month. |
| 1 week to 1 month | 37 | |
| 1 month to 3 months | 13 | 52 cases more than a month and less than a year. |
| 3 months to 6 „ | 21 | |
| 6 „ 1 year | 18 | |
| 1 year to 2 years | 18 | 53 cases more than a year. |
| 2 years to 3 „ | 6 | |
| 3 „ 5 „ | 7 | |
| Over 5 years | 22 | |

viz.:

| | |
|---|---|
| 6 years | 3 cases |
| 7 „ | 9 „ |
| 8 „ | 1 case |
| 10 „ | 3 cases |
| 11 „ | 1 case |
| 14 „ | 1 „ |
| 16 „ | 1 „ |
| 18 „ | 2 cases |
| 20 „ | 1 case |

According to these figures, in one-third of the cases the second attack occurs within a month; in another third it is postponed until after a month, but occurs within a year, while in the remaining third more than a year elapses between the first and the second fit. I think it probable that these figures exaggerate the relative frequency of prolonged intervals, since in such cases the facts are more likely to be noted; but they serve at any rate to show that the risk of a second fit remains considerable for at least twelve months after the occurrence of the first fit, and that, although it rapidly falls after the second year, it does not practically disappear until after ten years have passed, while cases are occasionally met with in which a still longer period, 14, 16, 18, and even 20 years, elapses.

*Intervals between Subsequent Attacks.*—When the disease is confirmed, the intervals between the severe fits vary greatly, and the variation is still further increased by the effect of treatment.

In order to ascertain the proportion of cases in which the intervals are long or short, I have compared the intervals, at the time the patients first came under notice, in a series of 755 cases. In the majority the patients had not been under any treatment immediately before. In some cases the intervals were too irregular to permit of classification, but in 680 cases the intervals were fairly uniform, not exceeding the limits indicated in the divisions in the following table. Many patients have a series of fits and then an interval, the attacks thus occurring in groups. In this computation the intervals between the fits or groups of fits are taken into consideration, not those between the separate fits which occur together.

*Intervals between Fits in* 680 *Cases of Epilepsy.*

| | | Per cent. |
|---|---|---|
| Interval not exceeding 1 day | 88 | 12·9 |
| „ more than 1 day, not exceeding 1 week | 196 | 28·8 |
| „ „ 1 week „ 2 weeks | 111 | 16·3 |
| „ „ 2 weeks „ 1 month | 151 | 22·3 |
| „ „ 1 month „ 2 months | 75 | 11·1 |
| „ „ 2 months „ 4 „ | 43 | 6·2 |
| „ „ 4 „ „ 6 „ | 8 | 1·2 |
| „ „ 6 „ „ 12 „ | 8 | 1·2 |
| | 680 | 100·0 |

It thus appears that in more than three-quarters of the cases (80 per cent.) the intervals of freedom from fits did not exceed one month. In 57 per cent. the interval did not exceed two weeks, and in 40 per cent. it did not exceed one week, while in 12 per cent. the interval did not exceed one day. In only 8 per cent. was the interval greater than two months, and it exceeded four months in only 3 per cent. These proportions differ but little from those ascertained by Reynolds,[1] who found that of 64 cases analysed by him, the interval did not exceed a month in 51, or 80 per cent., and did not exceed a day in 11, or 15 per cent., while it exceeded two months in 5 cases, or 7·8 per cent.

A very large number of patients stated that they had their fits at intervals of one week (93 cases), a fortnight (72 cases), or a month (97 cases). Little weight can, however, be attached to these statements as evidence of a definite and regular periodicity. When such patients record the dates of the attacks, or are admitted into hospital, it is found that the intervals do not present the alleged correspondence with hebdomadal periods. It is evident that the statements are approximate only, and that the current divisions of time have suggested the definite assertions, rather than any terrestrial or lunar influences. A well-marked monthly periodicity is rarely observed except in the cases in women, not very common, in which attacks occur only at the periods of menstruation.

Many patients have attacks at intervals which vary beyond the limits given in the above table. This was the case in 75 patients, 11 per cent. of the whole number, in which the frequency of the attacks could be definitely ascertained. In these irregular cases the greatest interval was from five to forty times as long as the shortest interval. The latter varied from one day to three weeks, while the longer intervals varied, in different cases, from two weeks to two months. The minimum interval was less than a week in more than half the cases (45), and the maximum interval was more than a month in nearly three-quarters of the cases (51).

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[1] *Epilepsy*, &c. p. 148.

*Groups of Attacks.*—As already stated, the attacks may be isolated or grouped. The former is the more common, but the latter not infrequent. At periods which vary from one week to several months, series of fits occur, separated by intervals of a few hours, sometimes of a day. The number of attacks varies from two or three to twenty or more. Thus, one patient had a series of fifteen or seventeen fits every five days; another had a series of seven or eight fits in the course of a day or two, followed by freedom from fits for a week. The number of attacks occurring in each series often varies considerably in the same individual. Thus, one patient, at an interval of a week, would have sometimes only two fits, sometimes sixteen. Another patient had five or six fits every four weeks; a third had a series of seventeen fits, and then an interval of five days; in a fourth case several attacks occurred daily for a fortnight, and some months passed before another outbreak.

*Status Epilepticus.*—In the cases just described, the patient, as a rule, recovers consciousness in the intervals between the seizures. In rare instances a series of fits occurs in which the patient does not recover consciousness in the intervals, but, while in the post-epileptic sleep, another attack occurs. This has been termed the *status epilepticus* (*état de mal épileptique* of the French). It is a very grave condition. In its most severe form, which has been carefully studied by Bourneville,[1] the intervals between the fits become shorter, the coma deepens, the pulse and respiration become very frequent, and the temperature rises, it may be to 104°, 105°, or even 107°. Sometimes hemiplegia comes on after the condition has existed for several days. The patient may die in a state of collapse, death being apparently due to the violent and almost continuous convulsions, or, the fits ceasing, he may become delirious and present symptoms of meningitis, with rapid formation of bedsores, and may die in this stage. At any period the symptoms may lessen and the patient recover. A large proportion of the cases, however, end fatally. Fortunately this severe degree of the status epilepticus is very rare, at any rate out of asylums for the insane. No instance in which death occurred has come under my own observation, although I have seen many examples of a slighter degree of the condition, from which the patients have recovered.

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[1] *Etudes Cliniques et Thermométriques sur les Maladies du Système Nerveux*, 1873.

*Minor Attacks.*—The patients who suffer from minor attacks, with or without major seizures, are less numerous than those who suffer from severe attacks only. My notes on this point do not permit me to state the proportion in the series of cases analysed, but they support Reynolds' statement that the cases which present attacks of *petit mal* are less than half the whole series. The attacks of *petit mal* may occur alone, and constitute the disease; or they may occur together with severe attacks, or only with occasional attacks of hysteroid convulsion. The relative frequency of these combinations, in 250 cases in which attacks of *petit mal* occurred, is as follows :—

| | | | | |
|---|---|---|---|---|
| Both minor and major attacks | 174 | or | 70 | per cent. |
| Minor attacks only | 53 | „ | 21 | „ |
| Minor attacks with occasional hysteroid attacks | 23 | „ | 9 | „ |

*Intervals between Minor Fits.*—Minor attacks are, as a rule, very frequent in their recurrence. In more than half the cases they recur daily, as is shown by the following table of the recurrence in 100 cases, in which the interval could be ascertained with exactness :—

| | | |
|---|---|---|
| Attacks daily | 58 | cases |
| Interval two days | 4 | „ |
| „ three „ | 2 | „ |
| „ 'few' „ | 16 | „ |
| „ one week | 4 | „ |
| „ two weeks | 1 | „ |
| „ three „ | 1 | „ |
| „ four „ | 5 | „ |
| 'Infrequent' | 9 | „ |
| | 100 | „ |

In most cases the minor attacks recur far more frequently than the severe attacks in the same case. The frequency of the slight fits is sometimes very great. I have known as many as two hundred to occur daily. More frequently two or three occur daily, and the severe fits at intervals of one or two weeks. Occasionally, although minor attacks occur daily, the interval between the severe fits is considerable, two or three months. In sixteen of these cases with minor attacks (16 per cent.), although these occurred daily, no major attacks occurred at any time.

On the other hand, of the forty-eight cases in which the attacks occurred at intervals greater than one day, in all but one severe fits also occurred. The solitary exception was a case in which slight attacks occurred at intervals of three days, but the patient was not known to have had any severe fits. It thus appears that when attacks of *petit mal* alone exist, they are usually frequent, occurring daily, while, if the minor attacks occur less frequently, they are almost invariably associated with severe fits.

The rule that the minor attacks are more frequent than the severe is true of all cases in which the intervals between the minor attacks are less than two weeks. But cases are sometimes met with in which the severe attacks are frequent, occurring at intervals, for instance, of two, three, four, or seven days, and in which the attacks of *petit mal* occur only at much longer intervals. These cases are however rare, constituting not more than 7 per cent. of the cases with minor fits.

Occasionally there is a relation between the slight and the severe fits. The slight attacks occur only for a few days before, or, much less commonly, for a few days after, the severe seizures. In these cases the interval between the severe fits is usually several weeks. As already stated, the minor fits may occur alone at the commencement of the disease, and then, after months or years of slight seizures, the severer fits may occur. In the same way, if the severe attacks are arrested by treatment, the slight fits often continue.

*Time of Attacks.*—Another subject which deserves notice is the relative frequency with which fits occur by day or by night, using the terms as synonymous with the sleeping and the waking states. The investigation of this point in 840 cases has yielded the following conclusions:—The attacks occurred only, or almost only, at night in one-fifth (21 per cent.); only, or almost only, by day in rather more than two-fifths (43 per cent.) They occurred by both day and night in rather less than two-fifths (37 per cent.) Fits occurred in the night, partially or only, in rather more than half the cases (57 per cent.); by day, partially or only, in four-fifths of the cases (80 per cent.) In 1 per cent. the attacks occurred only while going to sleep; in ½ per cent. only on waking up out of sleep. In 5 per cent. the attacks occurred only in the early morning. A much larger number had their first attack in the early morning, and many of these patients were in the habit of early rising, a habit which, it thus appears, should be practised with caution by those predisposed to epilepsy. Between the cases with epileptic and those with hysteroid convulsions there was no difference in the frequency with which the attacks occurred in the day or in the night. The proportion, for instance, in which the attacks occurred exclusively, or almost exclusively, by night was, of epileptics, 21 per cent., and of cases with hysteroid attacks, 20 per cent. The occurrence of hysteroid attacks during sleep has been before alluded to (p. 145). In epileptics it is somewhat more common for the first fit to occur in the day than in the night—rather more than half commenced in the former, rather less than half in the latter. In hysteroid attacks it is much more common for the first fit to occur in the day than in the night. Two-thirds commenced in the day, one-third in the night. Nevertheless, in more than half these cases (56 per cent.) some fits subsequently occurred during the night—that is, during sleep.

When the first fit occurred in the day, the subsequent fits occurred only in the day in half the cases, only in the night in one-seventh, by both night and day in one-third. When the first fit occurred during the night, the subsequent fits occurred only at night in about two-fifths of the cases, by both day and night also in about two-fifths, and by day only in one-sixth.

If we may apply to these proportions the doctrine of probabilities, we may say that if a patient who has his first fit in the day has subsequent fits, the probability is as six to one that some of his attacks will occur in the day; the probabilities are equal that his attacks will, or will not, be confined to the day; and they are equal that he will, or will not, have some attacks in the night; while the probability that he will have attacks only in the night is but one in seven. If a patient who has his first fit in the night has subsequent attacks, the probabilities are equal that these also will occur only by night, and that they will occur both by night and by day, being in each case two in five. The probability is as five to one that they will not be confined to the day—that is, in favour of some fits occurring in the night.

Frequently fits which have recurred for a time in one condition, during sleep, waking, or both, change their time. The conclusions deducible regarding these are as follows:—(1) When fits which have occurred only by night commence to occur by day, they commonly continue also during the night. (2) When fits which have occurred only during the day occur during the night, they commonly cease by day. (3) Attacks which have occurred by day and night often cease to occur in the day and continue at night, but very rarely cease by night and continue by day.

*Relation of Attacks to Menstruation.*—The relation of attacks in women to menstruation is a subject on which various opinions have been expressed. The facts I have to offer are small in extent, the point having been investigated in sufficient detail in only 82 cases. In one-twelfth (7 cases) no attacks occurred at the time of menstruation;

in one-third (29 cases) there was no difference at these times; in more than half (46 cases) the attacks were worse at the monthly periods. Most frequently they were worse before the period (17 cases); next in frequency they were worse during the period (15 cases), and much less frequently after the period (4 cases). In 10 cases the time of the exacerbation was variable, it occurred either before, during, or after menstruation. In two cases the attacks were worse at the period soon after the patient became subject to fits, but not when the disease had existed for some years. In two cases fits never occurred at the periods when menstruation was regular, but they did when it was irregular.

# CHAPTER X.

## *PATHOLOGY.*

THE naked-eye appearance of the nerve-centres in idiopathic epilepsy is for the most part that of healthy organs. The slight opacity and thickening of the meninges in some cases of long duration, and the more distinct signs of meningitis sometimes seen after death in the status epilepticus, are apparently merely secondary changes. If the patient has died in a fit, the post-mortem appearances are those of the intense venous engorgement which is so conspicuous during life, and they differ little from those met with after ordinary asphyxia.

Great as is the aid which, during the last twenty years, the microscope has afforded in the investigation of the structural changes which underlie or constitute many diseases of the nervous system, it cannot be said to have thrown much light on the nature of idiopathic epilepsy. Of the minute histological changes which have been described, most, if not all, of those which are not common to many other diseases, are apparently secondary changes, the result and not the cause of the violent functional disturbance, or the effect of the repeated passive congestion to which the organs have been exposed. Such are the increased size of the blood-vessels of the medulla oblongata, described by Schroeder van der Kolk; the distension of the perivascular sheaths with pigment, the relics of former extravasations; and the yellow pigmentation of the nerve-cells of the medulla described by Voisin.

It is doubtful whether any greater significance is to be ascribed to the induration of the cornu Ammonis (*pes* *hippocampi*), to which so much weight has been attached by Meynert. Although the occasional occurrence of this change has been corroborated by several observers,[1] it has been found in only a few of the cases examined, and we are still ignorant whether it is, in some of these, a primary lesion, or is, in all, a secondary change. The association, by Sommer,[2] of the commencement of attacks by a sensory aura, with this change in a region near which we must look, according to Ferrier, for important sensory centres, is, as yet, suggestive only.[3] All physiological and pathological considerations render it improbable that the lesion has any direct relation to the process of convulsion. In the cases of epilepsy in which I have examined it, the cornu Ammonis was perfectly healthy, and in two cases in which the structure was diseased, the patients had never suffered from convulsion or epileptic symptoms. Nor can much weight be attached to the changes Echeverria[4] has described in the sympathetic ganglia. It is difficult to conceive that a disease which may exist for fifty years without the slightest other symptom of disordered action of the sympathetic, should be due to conspicuous alterations in these ganglia.

In considering, then, the pathology of epilepsy, we must seek other evidence as to its nature than that which is afforded by the pathological anatomy of the idiopathic disease, and must draw our inferences from the morbid changes in organic disease attended by convulsion, from the results of experiment, and from the facts ascertained by the clinical study of the disease.

It has been already pointed out (p. 42) that the muscular spasm which constitutes the most conspicuous feature of the attack, must be regarded as due to the sudden violent action of nerve-cells—*i.e.* to the 'discharge' of grey matter; and that the sensations which the patient experiences before losing consciousness are due, directly or indirectly, to the same cause—to the commencement of the discharge. Premising this, the subject of the pathology of epilepsy resolves itself, in the main, into four questions: What is the seat of the discharge which thus produces the symptoms of the fit? Is the seat of the discharge the seat of the disease? How far does such discharge explain all the symptoms of the attack? What is the nature of the morbid change which causes the discharge? Perhaps to no one of these questions can a conclusive answer be given. Nevertheless, these are facts which indicate the direction in which an answer is to be sought, and, what is equally important, the direction in which we must not seek it too exclusively.

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[1] The elder Foville, Hemkes, Pfleger, Sommer.

[2] *Archiv für Psychiatrie*, Bd. x. Heft 3.

[3] It has been pointed out by J. J. Putnam that the fact that this structure is commonly described as altered on both sides seems to militate against the theory of Sommer. (Supplementary volume to Ziemssen's *Cyclopædia*, 1881, p. 622.)

[4] *Epilepsy*, 1870, p. 111.

What is the seat of the primary discharge? What grey matter is it which thus suddenly stimulates the nerve-fibres and the muscles? It is hardly necessary to remark that the spinal cord acts only as a conductor, and that the overacting grey matter is to be sought for above it, within the skull. Regarding the encephalic masses, we have first the teaching of experiment. On the one hand the researches of Brown-Séquard and Kussmaul demonstrate that convulsions may take origin in the pons and medulla, since they may occur when all other parts of the brain have been removed. Nothnagel has further shown that there exist in the medulla oblongata, adjacent to the centres which regulate the respiratory movements and the state of the vessels, structures capable of giving rise, by their action, to general convulsions—'the convulsive centre.' On the other hand, of all regional diseases of the brain, lesions of the convolutions stand incomparably first as a cause of convulsions, and the experiments of Ferrier and Luciani[1] also demonstrate that irritation of the cortex in the motor region has the same effect. The teaching of experiment, then, is that both the cortex and the medulla may originate convulsions. The teaching of pathology is, as Wilks long ago insisted, that epileptiform convulsions have, in most cases, their origin at the surface of the brain. It may be doubted, however, whether the pathological facts alone, or in conjunction with experiment, quite warrant the conclusion that epilepsy is a disease of the cerebral cortex. Burdon-Sanderson's researches[1] make it probable that the convulsions which occur when the surface is irritated may depend on the discharge of motor centres more deeply seated, though connected with, and excitable from, the surface regions. So far as pathology and experiment go, these deeper structures, at the level of the corpus striatum, or perhaps lower still, may be the parts which discharge in epilepsy. The fact that lesions in certain regions of the cortex cause convulsions beginning in certain parts, as the arm or the leg, is not incompatible with this view, since discharges beginning in the lower centres may have quite the same definite forms as when they are excited from the surface. Physiological considerations make it probable that the arrangement of represented movements in the lower centres is on the same plan as in the higher. Nor does the affection of consciousness afford valid ground for the assumption that epilepsy is a disease of the cortex, since those who believe that the discharge originates in the medulla find an explanation of the loss of consciousness by assuming an extension of the discharge to the vasomotor centre, causing arterial spasm throughout the brain. Even if we admit, for a moment, that there is no proof of arterial spasm, and that the overaction of motor grey matter is the only certain element in the convulsions, such overaction may disturb the centres above as well as the centres below, and, as Robertson of Glasgow has suggested, the loss of consciousness may be the effect of an upward discharge, just as the muscular spasm is the effect of the downward discharge. Thus, neither pathology nor experiment enables us to exclude the lower encephalic centres as the seat of the primary discharge in epilepsy; and, in the light of all known pathological and experimental facts, the latest writer[1] of a systematic account of the disease, Nothnagel, can see no reason to modify the conclusion that the primary seat of the discharge—the seat of the disease—is the convulsive centre in the medulla oblongata.

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[1] *Rivista Sperimentale de Frenatria e Med. Leg.* iv. 1877, p. 617.

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[1] *Proceedings of the Royal Society*, vol. xxii., 1875, p. 368.

Pathology and experiment, then, fail to take us farther towards a definite conclusion, but from clinical observation we may gain other important information. At this point the study of the modes of onset of convulsions becomes of the highest value. The commencement in the limbs helps us but little, because, as I have said, movements in the lower centres are probably differentiated, if not as much, yet on the same plan, as in the convolutions. But it is different when the auræ referred to the special senses, or consisting in mental processes, are considered. We have seen that, of all the fits which we begin so deliberately as to allow the patients to be conscious of the onset, a special-sense warning is present in a fifth—that is, the discharge, as far as we can ascertain, commences in a special-sense centre. But these centres are certainly situated within the hemispheres, above the pons, and far above the 'convulsive centre.' Hence the conclusion seems inevitable that the discharge in such cases commences in the hemispheres. It is equally clear that an attack which begins with an intellectual aura, an idea, cannot commence with a discharge in the medulla oblongata. Such an attack we can only conceive as commencing in the highest of all the cerebral centres, that which constitutes, to use the phrase made current by Spencer and Jackson, the anatomical substratum of intellectual processes.

We may therefore conclude, with Hughlings Jackson, that the teaching of pathology, if inconclusive alone, is right in its indications, and that, in at least a large number of epileptic attacks, the process of the fit commences in the cerebral hemispheres. The conclusion thus reached derives additional strength from the experimental demonstrations, already referred to, that epileptiform convulsions may be excited by irritation of the motor region of the cortex, and also from the case of epilepsy recorded by Oebeke (see p. 86) in which the convulsions were arrested on one side by a lesion in the central ganglia of the opposite hemisphere. It is exceedingly probable that the attacks which begin deliberately commence in those cortical centres, which have been proved to possess functions the disturbance of which constitutes the first symptoms of the attack; and our present knowledge of the physiology of the brain makes it almost certain that this is the case when the warning of the attack is of such high specialisation as to indicate a psychical process. In cases in which the initial symptoms are less elaborate, spasm or ‘crude’ sensation, we cannot at present exclude altogether the central ganglia as the possible seat of the discharge.

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[1] In Ziemssen's *Cyclopædia*, vol. xiv.

If this is true, and most epileptic fits are to be regarded as the expression of a morbid process in the cerebral hemispheres, the question arises, are we not justified in assigning all idiopathic convulsions to this situation? Are there any facts to indicate that the centres of the medulla are ever the seat of the primary discharge in idiopathic epilepsy?

It is certain that convulsions may arise from these lower centres, since there exists in the medulla the ‘convulsive centre,’ by the agency of which they may still be produced in animals after the removal of the cerebral hemispheres. Moreover, in man, convulsions are caused, in rare cases, by diseases of the pons and medulla. These facts make it probable that, as Luciani has suggested, the medulla may play a secondary part in the production of the convulsions in epilepsy. But the clinical facts to suggest that the discharge ever commences in this situation in idiopathic epilepsy are, it must be confessed, scanty. Does the study of modes of onset help us in this particular? There is one group of auræ which may be regarded as supporting the view that the fits which they initiate take origin in the medulla. I refer to those which I have grouped as the pneumogastric auræ, consisting of epigastric sensations rising to the throat, choking, dyspnœa, palpitation. We know that the pneumogastric nucleus and respiratory centre are in close proximity to, and connection with, the convulsive centre, and it is readily conceivable that a commencing discharge in this situation may lead to disturbance of the respiratory and pneumogastric centres before it causes general convulsions. But on this point other facts must be taken into consideration. The phenomena of conscious sensation make it certain that the whole body is represented in the cerebral hemispheres. It is conceivable that when a discharge originates in certain parts of the hemispheres, the central structures connected with the vagus, or representing the respiratory processes, may be the seat of the first discharge, or may, at least, be the most sensitive to the commencing disturbance, and be the channel through which the consciousness is first impressed. We must remember that, as we have already seen, and as Hughlings Jackson has pointed out, these central representatives of the vagus are the most readily disturbed in emotion, such as fear. Thus the early affection of these centres, which we have just considered to be possible in a discharge originating in the medulla, is equally possible in a discharge originating higher up. Much weight cannot therefore be placed on the pneumogastric aura as evidence that the discharge originates in the medulla. This conclusion is supported by cases which show that this aura is sometimes felt in attacks which certainly commence in the hemispheres. As was stated in the last lecture, this pneumogastric aura may be associated with special-sense warnings even of high elaboration, and an instance of this is the case which I related at length on p. 67.

We are probably not justified in concluding that attacks never arise in the medulla. The extremely varied character of auræ, referred as they are to every part of the organism, shows that the discharge may commence in any part of a very wide region, perhaps the whole of the brain in which sensori-motor processes are represented. The cases are, however, very few which afford any clinical evidence that the discharge commences in the medulla. It is possible that, in cases of reflex convulsions, the process occurs in this situation. Almost the only example of idiopathic epilepsy which has come under my notice in which the symptoms suggested that the discharge originated there was the patient whose case is mentioned on p. 91, in which passive movements of the trunk, slight flexion of the spine, invariably caused an epileptic fit, attended by loss of consciousness. Although there was no epileptogenic zone, the attacks remind us of Brown-Séquard's epileptic guinea-pigs, in which the convulsions certainly did not originate above the pons.

The conclusion, then, is that the convulsions in idiopathic epilepsy may probably be due to the discharge of any of the grey matter of the encephalon, which subserves sensori-motor processes. This brings us, however, to the second question: Is the seat of the primary discharge the seat of the disease? If, by such discharge, all the symptoms can be accounted for, we have no need to go further, and the theory that any other pathological process underlies the phenomena must rest upon its independent evidence. To learn, therefore, whether the seat of the discharge can be regarded as the seat of the disease, we must consider, first, whether there are any symptoms for which the discharge of grey matter, which causes the aura and the convulsion, cannot account; and, secondly, whether there is direct evidence of any other pathological process.

It has been supposed that the motor discharge, especially if it occur in a low centre, will not account for the loss of consciousness. We may doubt whether this assumption is justifiable with regard to any part of the brain, even to the medulla oblongata. A sudden lesion of the brain, in almost any part, will cause transient loss of consciousness. All parts of the brain are intimately connected; the whole must be sensitive to a change in any region. The maintenance of consciousness is its highest function, the first to be interfered with by any disturbance, such as results from a discharge. If the discharge originates in some cases in the lower centres, its upward effect may, as already suggested, cause a disturbance in the highest centres with which consciousness is incompatible. For the attacks characterised by loss of consciousness only, the theory held by Hughlings Jackson, that the morbid process affects the highest centres only, seems an adequate explanation, and is rendered probable by the fact that such attacks are often attended by sensations of a highly specialised character. In the case of the lower centres, it seems possible that a *slight* discharge in some situations may affect the centres above, and not the centres below—may impair consciousness, and not cause convulsion.

Thus it seems unnecessary, for the explanation of loss of consciousness, to assume the occurrence of any other pathological process than the discharge which causes the convulsive phenomena. As far as I am aware, the only other symptom which has been held to indicate that there is more than this discharge is the pallor of face which is sometimes conspicuous. But the peripheral vaso-motor nerves are known to be influenced, in a very ready way, by the stimulation of other nerve-structures. That pallor of face may *result* from cerebral action is proved by the effect of emotion, whatever be the mechanism by which that effect is produced. An injury to the brain of a frog will cause the arteries even of the foot to contract. Hence it is readily intelligible, and even probable, that pallor of face may be, as Hughlings Jackson[1] has suggested, an *effect* of the discharge which causes the convulsion.

If then it is unnecessary, in order to explain all the symptoms of attacks of epilepsy, to go beyond the discharge of which we have certain evidence, we must ask what direct evidence there is of the occurrence of any other morbid process. According to current theory, another element plays an important part in producing the phenomena of attacks—vaso-motor spasm. It has been held that the discharge itself, though originating in the hemispheres, is due to vaso-motor spasm affecting particular arteries, and exciting the action by causing local cerebral anæmia. This view, which regards epilepsy as simply a disease of the vaso-motor centre in the medulla, is now held by few. It is unnecessary to discuss it separately, since the evidence on which it rests will be considered in speaking of a second view, much more widely held, that, although the convulsion depends on the discharge of motor or convulsive centres in the medulla, the loss of consciousness is the result of arterial spasm in the hemispheres due to the action of the vaso-motor centre in the medulla. A third theory of vaso-motor influence is that put forward by Hughlings Jackson, that the local discharge in the brain excites, at the spot, arterial contraction, and that this determines the *spread* of the discharge.

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[1] *Brit. Med. Journal*, Jan. 25, 1879, p. 111.

What, then, is the direct evidence that vaso-motor spasm in the brain causes loss of consciousness, or any of the symptoms of the attacks? The evidence may be put, if somewhat baldly, yet I think accurately, thus:—Epileptic attacks are accompanied, at the onset, by pallor of face. Pallor—that is, anæmia—of face is evidence of anæmia of brain. Anæmia of brain, as Kussmaul and Tenner have shown, may cause loss of consciousness and general convulsions. The arteries of the meninges (and probably also of the brain substance) may be made to contract by irritation of the sympathetic in the neck. Therefore loss of consciousness in epilepsy is due to anæmia of brain, the result of vaso-motor spasm; and, some would add, the convulsions themselves are due to the same cause.

The gaps between several of the steps of this reasoning will doubtless seem to many, as they do to me, considerable. But the first two statements are themselves of doubtful truth. I have pointed out that epileptic attacks are often accompanied by no pallor of face, and this is true most conspicuously of many attacks of *petit mal.* Further, the assumption that pallor—*i.e.* vaso-motor spasm—of the face is necessary evidence of vaso-motor spasm in the brain seems scarcely justifiable. Surface vessels have no necessary correspondence in their condition with those of deeper structures. We do not infer that the brain is congested whenever the face blushes, and why should we assume that the brain becomes anæmic whenever the face becomes pale? We have seen that it is conceivable, and even probable, that the discharge in the brain may cause a reflex contraction of the peripheral vessels. In cases in which the pallor of face is apparently the result of cardiac failure, it is probable that this is the effect of the commencing discharge, and not its cause. Convulsion is not part of the ordinary phenomena of cardiac syncope. On the other hand, as the discharge often impresses the consciousness first through the pneumogastric centres, it is probable that it may in some cases influence the periphery first by the same channel. That such cardiac failure is not the *ordinary* cause of loss of consciousness in epilepsy, a very small amount of clinical observation will demonstrate, since such loss of consciousness may occur without either failure of the pulse or pallor of the face.

Thus between the fact that profound anæmia of the brain and medulla oblongata will cause both loss of consciousness and convulsion, and the theory that these are commonly so caused in epilepsy, there is a gulf over which no bridge has yet been placed.[1] It is a gulf, moreover, which is widened by every addition to our knowledge of the clinical history of the disease.

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[1] 'Although experimental investigation has thrown much light upon the dark places of experimental pathology, it has brought with it some confusion into the region of practical medicine. There are analogies and close relations between the convulsive phenomena of rabbits which are bled to death, of guinea-pigs with their spinal cords half sundered, and the convulsive paroxysms of epilepsy; but the cases are not identical, and although experiment may elucidate the mechanism of the attacks—some parts of which may be the same in the two cases—it may leave untouched the real pathology of the disease, the clinical history of which is peculiar to itself and more or less widely different from those of the several paroxysms artificially induced.'—Reynolds; *Epilepsy*, p. 250.

One vaso-motor theory remains for discussion—that which assumes that a local discharge in the brain excites, at the spot, reflex vaso-motor spasm, and that this determines the spread of the discharge. This theory, I would venture to suggest, is scarcely warranted by known facts, or needed to explain them. It is unneeded, because the anatomical relations of known centres already afford us the means of explaining many facts in the progress of auræ, and justify us in the belief that the explanation of the others, still mysterious, will be found in similar relations of other centres, and especially of those which subserve sensation. The theory seems scarcely warranted, because in all organs, as far as we know, functional activity causes reflex dilatation, not contraction, of their vessels; and the experiments of Ferrier have proved that this is as true in the brain as elsewhere.

One other point on the current theory of epilepsy may be briefly noticed—that which refers the final clonic spasm to a cause different from that which excites the initial tonic spasm. The clonic spasm is ascribed to the irritation of the medulla by the venous blood. It has, however, been pointed out (p. 77) that there is no abrupt transition from the one form of spasm to the other. The clonic spasm is developed by interruptions in the tonic spasm, and as the fit ceases the muscular contractions do not become slighter, but less frequent. The clonic stage certainly corresponds to the cyanosis which results from interference with respiration, but if we recognise the continuity of the tonic and clonic spasm, we can hardly attribute the two to totally different causes. Observation of fits suggests that the venous congestion does produce the clonic spasm, not, however, by direct excitation, but, as Axenfeld and A. Foville asserted, by interrupting and ultimately arresting the spasm which before existed. The fixation of the chest in an epileptic fit simply does in an intense manner that which we do in a slighter manner when we arrest an hysterical convulsion by closing the mouth and nose. There is nothing inconsistent in a certain degree of anoxæmia arresting spasm, and a more profound degree, as in actual asphyxia, exciting it. Analogous difference is seen in the action of many stimuli. The view that the tonic spasm is only clonic spasm compressed, that the clonic spasm is only the tonic spasm spread out, is supported by the fact that in attacks which begin slowly the slight initial spasm is almost always clonic, and as the discharge spreads and becomes more intense the spasm becomes tonic. In partial epileptiform seizures the spasm is often clonic throughout.

The conclusion, then, is that all the phenomena of the fits of idiopathic epilepsy may be explained by the discharge of grey matter; that the hypothesis of vascular spasm is as unneeded as it is unproved; that there are no facts to warrant us in seeking the seat of the disease elsewhere than in the grey matter in which the discharge commences; that this is in most cases within the cerebral hemispheres, probably often in the cerebral cortex, although possibly in some instances lower down, even in the medulla oblongata; that epilepsy is thus a disease of grey matter, and has not any uniform seat. It is a disease of tissue, not of structure.

The last question is, What is the nature of the tissue change? Can we form any opinion as to the character of the alteration in the grey matter which permits its sudden explosive discharge? This problem is essentially the same, whatever theory of epilepsy is adopted. We must assume an instability of nerve-cells somewhere, in the vasomotor centre or the convulsive centre, if we do not in the cerebral hemispheres. The violent muscular contractions imply a corresponding overaction of the nerve-cells, and it has seemed to some, as Hughlings Jackson, that such repeated overaction can hardly be other than an indication of increased functional activity—of increased, if abnormal, nutrition. On the other hand, others, as Radcliffe, have maintained that the general conditions of epileptics and the phenomena of epilepsy suggest impairment of functional power, and that a symptom so abnormal as an epileptic fit can hardly result from normal nutrition, to whatever excess it may be carried.

Modern physiology teaches us, I think, in what direction an answer is to be sought. It is necessary, in the first place, to avoid too direct an inference from the outward manifestation to the internal process. No one probably now imagines that the nerve-force which sets a muscle in action forms part of the force liberated by the muscle; and, although the two are no doubt proportioned, the difference between their degree may be as great as between the energy liberated by an electrical spark and by a charge of gunpowder which it fires. We may easily overrate the amount of nerve-force needed to cause violent muscular action, and may also overrate the relation which the nerve-force bears to the potential energy of the cells concerned in its production.

Every nerve-cell may be described as a storehouse of latent energy, and may be compared, by analogy, to a charged Leyden jar, or, what is more convenient for our present purpose, to a bent spring. The energy of the spring is due to the force which has bent it, and depends on the resistance which keeps it bent. The recognition of a resistance to action in nerve-cells, and of the part which it plays in normal processes, is a most important step in modern physiological thought. Of the nature of the resistance we can form perhaps no idea, but its existence in nerve-cells may be taken as hardly needing proof; we are unable to conceive of their action without it, and the idea has underlain such expressions as 'nerve-tension,' and the like, which have been long employed. The fact is probably only the highest instance of a series of relations which runs through the whole of organic nature. The formation of every organic compound, for instance, consists in, so to speak, the bending back of the spring of chemical affinity in a process by which force is held latent by some unknown resistance.

The application of this mode of thought to the morbid action of the nervous system promises to yield clearer views of many problems of disease, as it already has elucidated many of the processes of health. The state of a nerve-cell, whether at rest or in action, may be regarded as depending on the proportion which its tendency to liberate energy bears to the resistance within it. The action of a nerve-cell may depend on the production of force within it being increased in excess of the resistance, or on the resistance being unduly lessened. The cessation of its action may depend on the tendency to liberate energy being diminished or on the resistance being increased. It is highly probable that all the phenomena of 'inhibition' depend on increase of resistance by the action of one cell upon another. It is, indeed, conceivable that the interaction of the nerve-cells of the brain may be such that their mutual influence is a balanced resistance, and the amount of force in the brain, nascent but restrained, may be infinitely greater than any manifestations of energy which we perceive could possibly suggest. We may, perhaps, ultimately learn to regard the effect of stimuli in developing nerve-action not after the analogy of the mechanism of a piano, each blow upon a key directly exciting the sound, but rather after that of an organ, in which the movement of a key merely lessens the resistance to the action of a pent-up force.[1]

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[1] The application of the doctrine of 'resistance' to the explanation of the physiology of the nervous system is due, in a large degree, to Wundt in Germany, and, in this country, to Michael Foster, who has made it current by his masterly application of it in his *Manual of Physiology*. The principle was, however, clearly enunciated in 1873 by Handfield Jones in the following passage:

'In the construction of a motor nerve-centre, it is requisite to create an apparatus which shall be always ready to evolve force, but which shall not do so spontaneously—not without the application of a stimulus of some kind or other, physical or mental. Now, the peculiarity of nerve-cells is that they possess these two qualities; they prepare material which, by undergoing oxidation, or in some other way, generates force; and yet they can prevent this material from so acting, although blood is circulating all round it charged with oxygen. . . . In states of spasm, this property of the nerve-cell is lost or much impaired.'—'Lecture on Paralysis Agitans,' *Brit. Med. Journal*, 1873, vol. i. p. 221.

Ringer has very clearly applied the theory to the explanation of tetanus (*Med. Chir. Trans.* 1876).

Do not, I would ask, the phenomena of epilepsy best accord with the theory that the unstable equilibrium of grey matter, its tendency to discharge, depends on instability of resistance rather than on any primary change in the energy-producing action of the cells? When loss of blood developes general convulsions, by its action on an otherwise healthy nerve-centre, can we conceive, I would ask, as Radcliffe has asked before, that it acts by increasing the production of nerve-force? It appears far more intelligible to regard its effect as a sudden diminution of resistance, by which the pent-up nerve-force is suddenly released, the nascent energy developed. The arrest, by the ligature, of fits which begin deliberately seems to point in the same direction. A strong peripheral impression causes such a change in the grey matter to which the discharge is advancing, that the discharge is arrested there, and passes no further. We can hardly conceive the effect of the impression except as causing a local increase of resistance, so that the liberation of energy ceases at the spot, can no longer occur. Once arrested in this way, it is arrested for a considerable time. If the discharge depended on a primary increase in the energy-producing action of the cells, we should expect it to recur when the ligature was removed. It does not. I have already mentioned the case of a patient who has, for a long time, arrested every fit, commencing in the hand, by a ligature above the elbow. At first, if he did not apply the ligature, the attack went on into a general convulsion. He now finds that the sensation, beginning as before, stops, of its own accord, at the spot at which the ligature used to be applied, and never goes on to a fit. According to the view I have suggested, the repeated increase of resistance has led to a corresponding permanent change, resting on an alteration of nutrition, so that the unstable grey matter is limited by a zone of greater stability, where the discharge is arrested.[1]

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[1] That variations in tissue-resistance may influence the *spread* of a discharge which has already commenced has been recognised by Hughlings

Analogy suggests that the internal resistance to action is a higher function of the nerve-cells than the mere transformation of energy. The control is a higher function than the production of the force. Hence the fact that there is an apparent overaction becomes consistent with the theory that there is imperfect nutrition.

The discussion of the real nature of these influences—the liberation of energy, and the resistance to action—how far they are ultimately to be referred to vital, chemical, or electrical changes, is beyond the scope of this work. The recognition of one point only appears to me essential—that nutritive changes may underlie them, and that from them nutritive changes may result. Although the production of nerve-force may not be primarily excessive, it may become increased by the increased demand upon it by the repeated discharges. Then what may be called a state of secondary excessive nutrition may occur, but a nutrition which increases the energy-producing and not the energy-restraining power; and this may be the reason why the disease, after a time, becomes so hard to check.

Whatever opinion may be formed regarding the parts played by the energy-controlling and energy-producing elements, respectively, in the production of the phenomena of the 'discharge,' and thus constituting the ultimate lesion in epilepsy, there is an important class of epileptic phenomena which, it seems to me, can be referred to but one of these factors. We have seen that minor attacks may, in some cases, be characterised, not by

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Jackson. 'The discharge of highly unstable cells, constituting the primary discharge, leads to the secondary discharge of healthy cells in other centres (collateral or lower) with which there is an anatomical connection by fibres; the degree and width of the secondary discharges varying according both as the force of the currents developed by the primary discharge, and the resistance opposed by fibres and by cells of the healthy centres, varies. We must not forget the resistance offered to local discharges in our consideration of their effects. The "discharging lesion" may be likened to a fulminate which overcomes the resistance of less unstable compounds.'—*Brit. Med. Jour.*, Jan. 11, 1879.

sudden muscular spasm, but by sudden muscular weakness. The liberation of energy in the motor cells is suddenly diminished, instead of being increased, so that they no longer act, as before, on a given volitional stimulus, or do not act to the same degree. That the energy-producing power of the cells is for a time arrested, and then suddenly restored to its normal state, is an explanation of this condition which is scarcely conceivable. But that the resistance to the liberation of force in the cells is suddenly increased, that the cells are 'inhibited,' is an explanation not only conceivable, but in perfect harmony with our knowledge of the part played by inhibition in physiological nervous processes, and with the only received explanation of the nature of such arrest of action. We have seen that not only in the centres for the limbs, but also in the visual and auditory centres, such inhibition must be assumed in explanation of the phenomena of some attacks. We have seen, too, that discharge and inhibition seem to represent two opposite conditions of a centre—conditions which, in partial degree, may coexist, struggling, as it were, for the mastery, and the one or the other prevailing (pp. 64, 104). But if the inhibition can be regarded only as a sudden increase of resistance, it becomes in high degree probable that the discharge consists in a sudden decrease of resistance; and thus the view which, as we have seen, other facts suggest, is rendered still more probable.

Inhibition, the sudden increase of resistance, we know from the facts of physiology, to be usually the result of an action on the nerve-cells from outside them, the result of the functional activity of other nerve-cells. The phenomena of epilepsy make it probable that in disease this may also be the case. We have seen, for instance, that such inhibition of the motor centres for a limb may be the result of a discharge in the associated sensory centres, as in the attacks in which a painful sensation in a limb is accompanied by inability to move it. We do not yet know whether a sudden increase in resistance may arise primarily in a centre apart from an influence from without, but there is nothing inconceivable in the idea, and it is rendered probable by some epileptic phenomena, *e.g.* the occurrence of sudden weakness in a limb, as a minor attack, without any sensory discharge, or the occurrence of sudden loss of sight, before any other symptom, as the prelude to an attack. An instability of resistance may be conceivably manifested by a sudden abnormal increase as well as by a sudden decrease of that resistance.

If we accept, as the probable explanation of loss of consciousness in cases of discharge of the (relatively) lower cerebral centres, the view of Robertson that it is due to the influence of that discharge on the highest, that influence may readily be conceived as causing an increased resistance, an inhibition of the 'anatomical substrata of consciousness,' analogous to the inhibition of a motor centre. It is equally possible that some minor attacks, characterised by loss of consciousness only, may be due, not to a discharge of the highest centres, from a sudden lowering of resistance, but to an arrest of action by a sudden increase of resistance, a process the converse of that which constitutes a discharge.

The phenomena of hysteroid seizures seem to point to an instability of grey matter of different seat and perhaps different kind. The excessive violence of the muscular spasm in that disease is comparable only to the convulsions of epilepsy and tetanus. The phenomena appear due to the more deliberate and sustained overaction of centres which are concerned in specialised, coordinated movements, in emotions, and also in intellectual processes. The fact that these seizures may succeed attacks of epileptic *petit mal* has been regarded by Hughlings Jackson as evidence that the hysteroid convulsion (like the automatic action, p. 118) is always due to the release of lower centres from the control of the higher by the temporary discharge of the latter. He has urged that an actual discharge of grey matter can only give rise to incoordinate 'brutal' effects. That coordinated convulsive action may succeed attacks of true epilepsy, is beyond question; but it is also beyond question that the coordinated phenomena are not always preceded by epileptic symptoms, and, on the other hand, it seems certain that coordinated movements may result from a true discharge such as occurs in epilepsy. Evidence of this has been adduced at a preceding page (176).

It has been already suggested that even for the cases in which this symptom succeeds a true epileptic seizure the hypothesis of 'loss of control' scarcely affords an entirely adequate explanation. We can discern no difference between epileptiform attacks which are, and those which are not, followed by such symptoms. Hence their occurrence must depend upon some other condition, a condition pertaining to the nervous organisation of the individual, and not to the form of attack. This is supported by the fact that cases, in which epileptic attacks are succeeded by hysteroid convulsion, present preponderant characteristics in age, sex, causation, and conditions under which the fit occurs. In these respects the patients resemble those in whom such attacks of hysteroid character occur without any preceding epileptoid symptoms. These facts make it probable that there is in these epileptics, in addition to the instability of nerve-tissue which causes epilepsy, also the instability, whatever it may be, which gives rise to hysteroid attacks. 'Loss of control' may be the immediate mechanism by which this instability is thrown into operation, but will not alone account for its existence, and is no necessary condition for its manifestation. Nevertheless, in some patients the hysteroid tendency may be so slight that it never manifests itself spontaneously — never except during the post-epileptic state.

Such forms of attack, in which the convulsive phenomena are violent in degree and disorderly in sequence, are in the lowest grade of coordinated phenomena, while the highest are those forms of involuntary action which we call automatic. In these, complex actions are performed, sometimes with considerable skill, of which the patient is subsequently entirely unconscious. Hughlings Jackson has maintained that these are really post-epileptic phenomena. That they are commonly such, all must, I think, agree. The older view was that these were true epileptic events, the result of such changes in the brain as might, in another degree or part, constitute an ordinary epileptic attack. While recognising that they are commonly post-epileptic, I doubt whether the older view must be entirely discarded. Facts have been already mentioned (p. 176) which may make us hesitate to assent to the conclusion that the discharge of epilepsy must always cause incoordinate symptoms. What, for instance, is the vision of an old woman making ugly faces, which one patient had sometimes as the aura of a fit, sometimes as an attack of *petit mal*, but a coordinate discharge in—an automatic action of—the centre for visual ideas? The remarkable aura which I detailed at length (p. 67) suggests the same explanation, and so do many others that were alluded to. I have seen a patient suddenly, while being watched, without the slightest pallor, hesitation, or any symptom to indicate a previous 'discharge,' proceed to empty his pockets, take off his coat, or do some action which in him characterised, and I think constituted, his attacks of minor epilepsy.

With regard to these attacks of post-epileptic automatism, the same difficulties face us as in the case of the coordinated convulsion. We have the facts that in one individual attacks may always be followed by automatic action; and in another, similar attacks may never be so followed. This suggests that there is, underlying the phenomenon, a condition of the centres on which it depends. Were it not for this condition, the epileptic attack would not lead to it, and this condition may, in rare cases, cause its primary manifestation.

So too with regard to the violent attacks of automatic action which are termed 'epileptic mania,' and which, as we have seen, while usually post-epileptic, apparently sometimes precede epileptic fits, and sometimes occur apart from any recognisable fit.

# CHAPTER XI.

## *DIAGNOSIS.*

THE problem of the diagnosis of epilepsy includes (1) the recognition of the occurrence of attacks; (2) the distinction of the attacks from other paroxysmal affections with which they may be confounded; (3) the distinction of hysteroid from epileptic attacks; (4) if there is hysteroid convulsion, we have to ascertain whether it is simple or consecutive to an epileptic seizure; (5) if the convulsion is apparently epileptic, we have to consider whether it is due to reflex irritation, to blood-poisoning, or to organic brain disease, active or latent, and also whether it is simulated, before we can conclude that it is the manifestation of idiopathic epilepsy.

(1) *Recognition of the Occurrence of Attacks.*—Severe convulsive attacks are such obtrusive phenomena that there is little difficulty in ascertaining their occurrence, except in the cases in which they come on only when the patient is asleep. If the patient is not awakened by the commencing attack, and sleeps on when it is over, the convulsion is an event of which he may be entirely unaware. Usually, however, there are some subsequent indications of the attack. The tongue is bitten or sore; there is a little blood upon the pillow; or there are small ecchymoses upon the face of the patient, or an extravasation beneath the conjunctiva. I have known a patient to have convulsive attacks at night every few months for eighteen years, without being aware of their occurrence.

His tongue was occasionally found bitten in the morning, but the significance of this was not suspected, and the patient took no notice of it, until an attack occurred in the day-time. In Trousseau's case, already alluded to, nocturnal fits were only discovered by a dislocation of the shoulder; and the nature of this, from its occurrence during sleep, was at first mistaken.

Minor attacks are often unrecognised by patients or their friends. This is not because they are unnoticed, for since they occur in the day, they rarely escape observation, but it is because little importance is attached to them on account of their slighter character. It is usually sufficient to be aware of their common forms, and to enquire for these, to learn their occurrence.

(2) *Distinction of Attacks from other Paroxysmal Symptoms with which they may be confounded.*—Severe fits are so characteristic that their nature is usually at once recognised. It is very different with the minor attacks, the nature of which is very often misunderstood by patients and their friends, and sometimes even by the medical adviser. The simplest form, characterised by transient loss of consciousness, lasting for a moment, and passing away, to leave the patient just as before, is constantly regarded as a faint, signifying no more than an ordinary syncopal attack. The form which is characterised by a sudden start, or the dropping of what is in the hand, is attributed to 'nervousness,' and the attacks of transient giddiness are ascribed to a deranged stomach.

*Syncope.*—The distinction from fainting attacks rests, first, on the absence of obvious exciting influences. Syncopal attacks occur in weakly persons, and under the conditions of mental emotion, over-exertion, heated rooms, &c. The minor attacks of epilepsy occur in strong as well as in weakly persons, and come on at all times, and in all conditions, when the person is sitting still, as well as on other occasions. In epilepsy the loss of consciousness is often quite sudden; in syncope a feeling of faintness, or sickness, with perspiration, usually precedes the unconsciousness. But the absence of a sense of faintness is of greater value as an indication of the epileptic nature of the attack, than is its presence as evidence that the attack is syncopal, since some minor epileptic seizures are preceded by an aura which is described in similar terms. In epilepsy there is often also some other aura, such as those already described at length. Some of these, as the epigastric sensation, a visual sensation, or a sudden and peculiar mental feeling (*e.g.* a sudden sense of 'strangeness'), do not occur in syncope, and so are of considerable diagnostic importance. Giddiness, if definite vertigo, also suggests epilepsy rather than syncope. But indefinite 'dizziness,' palpitation of the heart, nausea, cephalic sensations other than pain, and sudden failure of sight, are of little diagnostic significance. The stage of complete loss of consciousness is much briefer in epilepsy than in syncope, and micturition, which may occur in the former, is unknown in the latter. Consciousness is often regained more suddenly in epilepsy than it ever is in syncope. It may be gone and back again in a second or two, and the patient be only aware of what has happened by finding that persons around are looking at him with surprise and alarm, or by discovering that something has happened which he cannot account for. A gentleman, for instance, while dressing, and standing by the side of his bath, suddenly found that his clothes were wet. He had had an attack of *petit mal* and had fallen into the bath, and got out again before he recovered perfect consciousness. If the patient's normal condition is slowly regained, there is physical faintness in syncope, and mental confusion in epilepsy. After the latter, some automatic action may be performed, a phenomenon never observed in cases of simple syncope. In the vast majority of cases, attention to the suddenness of loss of consciousness, or to the occurrence of an aura other than the sensations which precede syncope, with the circumstances in which the attack occurred, will generally enable the diagnosis to be made without difficulty. In a large number of cases of minor epilepsy the patient has other, more severe seizures, which assist the diagnosis.

*Vertigo.*—The attacks of minor epilepsy which are characterised by vertigo have to be distinguished from other forms of sudden giddiness. In some cases of epilepsy, vertigo is the only symptom of which the patient is conscious, and may be the only symptom of which we can obtain any account, although the vertigo is usually succeeded by transient loss of consciousness, obvious to others, but unknown to the patient himself. Other forms of vertigo, to whatever due, are rarely attended by loss of consciousness, and are usually followed by long-continued giddiness, which persists while the patient is recovering. In epilepsy there is no consecutive giddiness; the patient is at once well, or merely presents mental dulness for a few minutes. In epilepsy the giddiness may be associated with some other warning, as the epigastric sensation, which is absent in other forms of vertigo; and lastly, in epilepsy we often have the guiding indication of more severe attacks.

The form of vertigo most likely to give rise to diagnostic difficulties is auditory-nerve vertigo, 'Menière's disease,' the giddiness which is dependent on deranged function of that part of the auditory nerve which supplies the semicircular canals, and conveys information as to the position of the body—the 'space-nerve' of Cyon. Attacks of giddiness may come on in this condition as suddenly as in epilepsy, and in them the patient may fall, but they are never so brief. There is scarcely ever loss of consciousness, and the giddiness passes off slowly, and often incompletely; more or less giddiness persisting, with occasional slight exacerbations, for hours, days, or weeks. Other diagnostic indications are afforded by the distinctive symptoms of the auditory-nerve form—noise in the ear, which may not be more intense before a paroxysm; deafness, of which the patient may be quite unaware, since the loss may be confined to the perception of sounds conducted through the bone, and even to the appreciation of short high-pitched notes, such as those of a watch. Attacks of auditory-nerve vertigo are usually followed by vomiting, while this is comparatively rare after epileptic seizures. Paroxysms of auditory vertigo may often be brought on by sudden movements, and even by passive sudden rotation of the head, while this has no effect in bringing on attacks of epilepsy.

The greatest diagnostic difficulty is presented by cases of epilepsy in which consciousness is first affected by disturbance in that part of the brain through which consciousness is affected in auditory vertigo. The equilibrial centre is intimately connected with the pneumogastric nerve, and hence occurs the vomiting which so frequently succeeds vertigo, to whatever due. We have seen (p. 205) that the central representation of the pneumogastric is very often that through which consciousness is first affected in epilepsy. It is therefore not surprising that in some patients the symptoms of epilepsy should bear a very close resemblance to those of auditory-nerve vertigo. An auditory and pneumogastric sensation may be combined with vertigo as the warning of an epileptic fit. This was the case, for instance, in a man aged 20 who had suffered for six months from attacks which commenced with a sensation at the epigastrium 'as if it were turning over and over,' followed by a 'confused feeling' in the head, making him take hold of some support to avoid falling. This was succeeded by a whistle in both ears like a 'distant railway whistle going through his head.' There was, in addition, a sensation of 'pins and needles' in both feet, which ascended the legs as far as the knees. Consciousness was lost in the fit. Here the defective equilibration and the sudden noise in the ears resembled auditory-nerve vertigo. But the fact that the epigastric sensation preceded the other phenomena, and the occurrence of an aura in the legs, showed that the attack was epileptic; and this was confirmed by the circumstance that although the patient had some minor attacks, consisting only of the aura and momentary loss of consciousness, he had other attacks, preceded by the same aura, in which there was severe convulsion. There was no defective hearing in this case.

It must be remembered that a patient may suffer from both epilepsy and auditory vertigo. There is nothing mutually exclusive in the two morbid states. Indeed, their concurrence is intelligible if we consider, as I think we must, that the occurrence of vertigo, *i.e.* the disturbance of the equilibrial centre, which is produced by the ear disease, is facilitated by any primary instability of the nerve-cells; and such instability may be part of the general defective nutrition of the brain of which the epilepsy is an expression, or may actually result from the repeated epileptic 'discharges.' Much care is of course necessary in the recognition of the presence of two diseases which have many symptoms in common, but in several cases which have come under my notice, the evidence of the coexistence of the two affections has been clear. A woman suffered from distinct epileptic fits from 47 to 53, when they ceased. At 63 she became liable to paroxysmal vertigo—a sensation of falling backwards and towards the right, without loss of consciousness, and followed by vomiting. There was complete loss of hearing through the bone (watch and tuning-fork). The attacks of giddiness were often induced by movement, and were produced by sudden rotation of the head to the right, and not by rotation to the left. After she had suffered from these attacks for a year, the convulsive fits recommenced, the vertiginous attacks continuing. Again, a girl of 28 had been epileptic for several years, the fits, many of which were witnessed, being severe. She also suffered from almost constant giddiness, worst after rising in the morning, and varied by severe paroxysms of vertigo, often produced by movement, in which she seemed to fall backwards and to the left. She had constant noise in the ears, and loss of hearing through the bone.

*Neuralgia.*—Cases of minor epilepsy of which the aura is a sudden pain in the head may be mistaken for neuralgia; but the occurrence of transient loss of consciousness after the sudden pain is sufficient for the diagnosis.

(3) *The Distinction of Hysteroid from Epileptic Attacks.* If a patient suffers from attacks of distinct convulsion, the next question is, are the attacks epileptic or hysteroid? If a fit can be witnessed, it is rarely that any diagnostic difficulty will present itself. As a rule, the characters of the convulsion are distinct enough. The violent tonic and shock-like clonic spasm of the typical epileptic fit, with its complete unconsciousness, cyanosis, and brief duration, are wholly unlike the wild coordinated movements, perverted consciousness, talking, and biting of the hysteroid seizure. The other distinctive characters of the latter are the long-continued tonic spasm, the opisthotonos, the quivering of the eyelids, the quick clonic spasm, often of a quasi-volitional character, and maintaining the same rapidity throughout, the rolling up of the eyeballs when exposed, and especially their convergence.

There are, however, some attacks regarding the nature of which doubt may be felt, although the attack is witnessed. These are the intermediate forms of seizure which are described on p. 176, especially those in which there are brief, frequently recurring attacks, with complete loss of consciousness and quick recovery, but in which the convulsion differs from that seen in epilepsy and resembles somewhat that of hysteroid attacks. In these cases the diagnosis must be based upon the general characters of the disease rather than on the features of the convulsion, especially on the suddenness with which the attack comes on and passes off, its occurrence independently of exciting circumstances, the general conditions of the patient, and the absence of other manifestations of hysteria. When, in these features, the disease resembles epilepsy, it is to be regarded as such in spite of the character of the spasm.

Similar doubt may be felt regarding the nature of attacks which consist only of tonic spasm. Such attacks may be either epileptic or hysteroid, and in each there may be an apparent loss of consciousness. Suddenness of onset is in favour (but not conclusive) of the attack being epileptic. The occurrence of attacks when the patient is alone is still stronger evidence. If the patient manifests preceding mental, and especially emotional, disturbance, if the fits are excited by emotion and cease on a change in the patient's 'moral atmosphere,' if they never occur when the patient is alone, and if they can be arrested by strong sensory impressions, as by faradisation of the skin or by closing the mouth and nose for a few seconds, they are probably hysteroid. The last indication is subject to the exception noted on p. 160. The effect of treatment also gives assistance in the diagnosis in doubtful cases, since immediate cessation when bromide is given is in favour of the disease being epilepsy.

When an attack has not been witnessed, and, as is the case in a large number of instances, we are dependent, for our knowledge of its characters, on the description which is given by the friends of the patient, often not the most intelligent of people, we have much more difficulty in forming a diagnosis. The aura may help us a little. If it is palpitation of the heart, a feeling of general malaise, or a sensation in both feet, there is some probability that the attack is hysteroid. A careful enquiry should be made as to the form of spasm, whether coordinated or epileptoid. Hysterical patients 'struggle,' but if the friends are asked for this character an affirmative reply will generally be given, whatever is the actual character of the fit. If, as is often the case, the necessary information can only be obtained by a leading question, it should be put alternatively—'Does the patient struggle or does she jerk in the fit?' Another indication which can generally be elicited is whether the patient has to be held down or merely prevented from hurting himself or herself. The former points to hysteria, the latter to epilepsy. Tongue-biting is another important indication, since hysterical patients (in this country) never bite the tongue, but they often try to bite other people, which should also be asked for. Micturition and talking in fits are other important indications. I have arranged these diagnostic points in the following table :—

*Diagnostic Characters of Epileptic and Hysterical Fits.*

| | EPILEPTIC. | HYSTEROID. |
|---|---|---|
| Apparent cause . | Absent. | Emotional disturbance. |
| Warning . . | Any, but especially unilateral or epigastric aura. | Palpitation, malaise, choking, bilateral foot aura. |
| Onset . . . | Commonly sudden. | Often gradual. |
| Scream . . | At onset. | During course. |
| Convulsion . . | Rigidity followed by 'jerking,' rarely rigidity alone. | Rigidity or 'struggling,' throwing limbs and head about. |
| Biting . . . | Tongue. | Lips, hands, or more often other people and things. |
| Micturition . . | Frequent. | Never. |
| Defecation . . | Occasional. | Never. |
| Talking . . | Never. | Frequent. |
| Duration . . | A few minutes. | Often half an hour or several hours. |
| Restraint . . | To prevent accident. | To control violence. |
| Termination. . | Spontaneous. | Spontaneous or artificial (water, &c.) |

(4) *Distinction of Simple from Consecutive Hysteroid Attacks.*—If there is evidence that the visible convulsion is hysteroid, we have to ascertain whether it is simple, or whether it is consecutive to an epileptic seizure. It must be remembered also that some patients who have epileptic attacks followed by hysteroid convulsion have, at other times, hysteroid attacks which do not succeed epileptic fits.

The recognition of an epileptic seizure preceding the hysteroid state is often very difficult if we have merely the friends' account of the compound attacks to guide us, and it is not always easy even when we can see an attack itself. When there is a severe epileptic convulsion, attended with tongue-biting, a hysteroid state succeeding it is clearly post-epileptic; but the slighter epileptic attacks have a less distinctive character. The pseudo-epileptic aspect of the initial stage of many pure hysteroid attacks (see p. 142) may closely simulate an initial slight epileptic seizure. Initial loss of consciousness for a few moments before any spasm, an aura such as is frequent in epileptic fits without hysteroid accompaniment, or a form of *petit mal* which is distinctly epileptoid, as putting the hands forward, stooping slightly, with a dull expression of countenance, micturition during the initial stage—these are all evidence that the initial attack is epileptic. But by far the most important evidence in such cases is that afforded by other attacks. Most patients who suffer from hysteroid attacks after epileptic seizures have at other times, or have had in the past, epileptic fits which are not so followed, and which may be ascertained by careful enquiry. These attacks may be major or minor, and when the latter they closely resemble the initial symptoms of the compound attacks, the nature of which is thus indicated.

If a patient has epileptic fits succeeded by hysteroid convulsion, extreme caution is necessary in diagnosing the occurrence of separate independent hysteroid seizures, since the probability in such a case is that these are really consecutive to slight epileptoid seizures. But in some cases the evidence of their independent nature is very strong, since they occur without any initial stage resembling an attack of *petit mal.* Or if there is an initial period of tonic rigidity resembling a minor attack of epilepsy, the recurrence of this from time to time during the attack, and its cessation when the fit is cut short by treatment, show that it is really hysteroid and not epileptic.

(5) *Nature of Epileptoid Attacks.*—The last question in diagnosis is: If the convulsion is recognised to be epileptic in character, is it the manifestation of idiopathic epilepsy, or is it due to reflex irritation, to toxæmia, or to organic brain disease, active or quiescent?

*Reflex Convulsions.*—Among the cases commonly regarded as the effect of reflex irritation are commonly included the convulsions which occur in young children at the time of the first dentition. In almost all such cases the evidence of defective osseous development which is termed 'rickets' may be recognised, and the convulsions are apparently the result of the extreme irritability of the nervous system which accompanies that diathetic state, This irritability leads to the occurrence of attacks of laryngismus stridulus and of general convulsions of every degree of severity. It is probable that, in many cases, such convulsions occur without any distinct peripheral irritation, since a mere emotion or a puff of air upon the face is often sufficient to produce them. They are usually referred to the backward dentition, to the irritation of the teeth. That such irritation may act as an exciting cause is undeniable, but it is certain that the backwardness of the teeth has not the significance, as a source of irritation, which is commonly assigned to it: the retarded dentition is simply part of the diathetic state, and is not necessarily attended with any irritation. In all cases, then, in which one or many convulsions occur in young children between six months and three years of age,—and in which the symptoms are not such (to be immediately described) as to suggest the existence of organic brain disease,—the evidence of rickets, and especially the beading of the ribs, should be carefully looked for, and will almost invariably be found. In the vast majority of cases the convulsions cease when the diathetic condition is removed, as by the administration of cod-liver oil and steel wine. But unless the nature of the cases is recognised and the diathetic state is treated, the convulsions may continue, and may persist as idiopathic epilepsy, or, after having occurred for two or three years, they may cease, to recur as epilepsy at the second dentition or at puberty. Moreover, it is especially common for such convulsions of rickets to persist in families in which there is an inherited tendency to epilepsy. This has been already considered in the chapter on 'Etiology' (p. 22). Hence we can only make an absolute distinction from epilepsy while the evidences of rickets are still present. When these have passed away, and the convulsions persist, they are not separable from idiopathic epilepsy.

Convulsions at the period of the second dentition may also be a result of the irritation involved in the shedding and growth of the teeth, and in all cases commencing at this period, the state of the teeth should be carefully attended to. But such convulsions are very rare except in persons who possess an inherited tendency to epilepsy, and too often they continue although the dental irritation is relieved. The diagnosis, then, of simple reflex convulsions must always be made with hesitation, unless the convulsions cease when the exciting cause has been removed; and the hesitation must be especially great in persons who exhibit an inherited tendency, or who have suffered from convulsions at the period of the first dentition.

Of other sources of reflex irritation which may cause convulsions, the most frequent are intestinal worms. They are of diagnostic importance chiefly in cases of recent fits, or in which only one convulsion has occurred. In such cases, especially in children, this cause should be borne in mind, and excluded by careful enquiry and, in most cases, by the administration of vermifuge remedies. Tape-worms and round worms are both occasional causes. The existence of the former may generally be ascertained without difficulty by the frequent passage of joints. It is doubtful whether thread-worms cause fits, in spite of the great irritation of which the patient is conscious. It is very rarely that convulsions can be distinctly traced to this cause, although it is so common. When convulsions excited by intestinal worms have continued for several months, they do not usually cease when the exciting cause is removed. The repeated fits have apparently induced a state of the nervous system similar to, perhaps in part identical with, that which exists in idiopathic epilepsy, from which these cases cannot then be separated.

The form of convulsion may sometimes lead to a suspicion that it is due to worms. It may be quite similar to the convulsions of idiopathic epilepsy, but frequently is slighter in degree, and consists only of tonic spasm. In the intervals between the fits convulsive starts are especially common.

Another occasional cause of reflex convulsions is the irritation of indigestible food. The fit is usually isolated, and occurs soon after the ingestion of the food, which is usually of such a character as to suggest itself at once as the cause. For instance, a medical student, whose bowels had been confined for a week, ate a hearty lunch of pickled mackerel, and drank with it a quantity of milk. He then had some beer, and went to a post-mortem examination, in the course of which he had a convulsive fit. There was no inherited tendency, and he had no fit before or afterwards.

The diagnosis that a fit which follows, and is no doubt excited by, injudicious food, is nothing more than a reflex convulsion, should be made with some caution, in children as well as in adults. It is doubtful whether such reflex convulsions occur, except in individuals who are predisposed to convulsion by a special state of the nervous system. This may be an inherited or acquired tendency to epilepsy, and the fit thus excited may be simply the first of a long course of attacks. Again, the predisposition which permits the excitant to be effective may be commencing organic disease of the brain, which, at the time, has caused no other symptom. I have seen several instances of this. For instance, a little girl, apparently in perfect health, swallowed a piece of slate-pencil, and had a fit a few hours later. She was admitted into University College Hospital, the slate-pencil expelled, and at the end of a week, having had no other fit, and being apparently well, was discharged. But she was admitted a fortnight later with headache, and paralysis of some cranial nerves, and optic neuritis. She was thought to have tubercular meningitis, but the course of the symptoms was slow, and when she died, about two months later, a large glioma of the pons Varolii was found. Again, another child, apparently in good health, had a convulsion after some scarcely judicious food. The child seemed well afterwards, but a few days subsequently had another fit, then became febrile, and a fortnight later the signs of tubercular meninigitis were distinct, and of this she died. In these patients, before the commencing disease in the brain caused other symptoms, it produced such an irritability of the nerve-centre that a slight cause led to a convulsive seizure. Thus the possibility that a fit, apparently excited by digestive derangement, may be the earliest expression of serious brain disease, or of the state of nervous system which constitutes epilepsy, must always be borne in mind.

*Toxæmic Convulsions.*—Of convulsions due to the presence in the blood of poisonous substances, only three forms are of importance in the diagnosis of epilepsy—those due to alcohol, to the blood state in kidney disease, and to lead. All these may cause convulsions of epileptoid character and chronic course, which are independent of any actual visible lesion in the brain.

The cause of the attacks which are due to alcohol is usually sufficiently obvious. They often recur during the state of intoxication. They are general convulsions, sometimes frequently repeated, which present nothing distinctive in their characters. The distinction from idiopathic epilepsy rests upon the ascertained relation of the attacks to their cause.

The convulsions which occur in acute uræmia rarely present any diagnostic difficulty, since the other signs of this blood state are usually sufficiently obtrusive. But it is important to remember that kidney disease may be attended by convulsions of chronic course recurring fits, indistinguishable in their characters from idiopathic epilepsy. They are, as far as I have seen, confined to cases of the granular kidney. The diagnosis rests, of course, on the detection of the signs of renal disease. In most cases the pulse is incompressible and the heart hypertrophied, and, since the heart and urine should be carefully examined in every case of epilepsy, the condition ought never to escape detection. I have met with this renal epilepsy in a young man, but it is much more frequent in persons beyond middle life, in whom the possibility of its existence should always be suspected.

Chronic convulsions from lead poisoning may bear the closest resemblance to the fits of idiopathic epilepsy. They are, as far as I have seen, always bilateral, and often begin with a visual aura. Their diagnosis rests on the detection of their cause. In all cases of epilepsy in persons whose occupations expose them to the risk of lead poisoning, the gums should be carefully examined. Not unfrequently the convulsions are associated with other nerve-symptoms, such as wrist-drop, due to the same cause. It must not be assumed, however, that in every case of epileptic fits in the subjects of lead poisoning the convulsions are due to this cause. I have twice met with the combination in cases in which the epilepsy commenced in early life, before the patient began to work with lead. Moreover, the subjects of chronic lead poisoning often also suffer from chronic renal disease, and it may be doubtful to which of these causes the fits are due, and in some cases it may reasonably be concluded that they are due to both.

The distinction of these cases of toxic convulsions from idiopathic epilepsy is rather etiological than clinical. The convulsions from lead and alcohol may persist after their original cause has ceased to operate, and, in such cases, the effect of that cause, and of the repeated convulsions, has apparently been to excite in the brain a pathological state similar to that which exists in simple epilepsy.

*Convulsions from Organic Brain Disease.*—The convulsions of idiopathic epilepsy have to be distinguished from those due to organic brain disease, active or inactive.

Convulsions often attend the onset of an acute cerebral lesion, or occur during the early stages of the lesion. They may occur, for instance, at the onset of cerebral hæmorrhage, or cerebral softening from vascular occlusion, but the lesion which causes the convulsions causes also other obtrusive symptoms, usually hemiplegia, which declare its existence. A convulsion beginning unilaterally may cause transient hemiplegia ('post-epileptic paralysis,' p. 99); and if the convulsion is the first from which the patient has suffered, a doubt may exist as to whether both convulsion and paralysis are not the result of an acute cerebral lesion. The weakness succeeding the hemiplegia very rarely amounts to complete paralysis; it is most marked in the limb (arm or leg) in which the fit commenced; and it passes away in the course of a few hours. When, on the other hand, the convulsion and paralysis are the result of an acute lesion, the paralysis is usually at first complete; it affects both arm and leg, and it scarcely ever passes away in the course of a few hours, usually lasting for days or weeks. In most cases, however, the patient has had other fits beginning unlaterally, and then, especially if the paralysis is incomplete, the diagnosis of post-epileptic hemiplegia may be made with confidence. In cases of softening from vascular obstruction, convulsions may occur a few days or a week after the onset, apparently from the adjacent irritation consequent on the strain upon the collateral vessels. In these cases, the convulsions succeed paralysis, and the diagnosis of their cause presents no difficulty.

Chronic active brain disease, especially tumour and chronic meningitis, may cause convulsions closely resembling those of idiopathic epilepsy. The convulsions usually begin locally, and affect one side first or only. In all cases of convulsions so commencing, and also in the case of those beginning with a visual or auditory aura, the probability that they are due to organic brain disease must be borne in mind, and careful search made for other symptoms. The mere fact of local commencement and deliberate march does not, alone, constitute evidence that there is organic brain disease (such as tumour or meningitis), since fits begin thus in idiopathic epilepsy not at all rarely. Moreover, the convulsions of organic disease may not begin locally; they may even begin with the common aura of idiopathic epilepsy, the epigastric-throat aura, as in a case of tumour of the pons (mentioned on p. 57). In such a case, however, organic disease would not be thought of unless there were other symptoms to suggest it, whereas, if fits begin locally, this should be the first idea.

The other symptoms which commonly concur with convulsion in organic disease are the following—headache felt not only after the fits, but during the intervals; hemiplegic weakness, persisting; paralysis of cranial nerves; vomiting; optic neuritis. The latter symptom is of extreme importance, since, as Hughlings Jackson long ago insisted, coexisting with convulsions of local commencement, it is all but pathognomonic of organic disease. Syphilis is so frequent a cause of such organic brain disease as causes chronic convulsions, that a history of it gives significance to symptoms otherwise doubtful. The mere occurrence of convulsions in a subject of constitutional syphilis does not prove the existence of organic disease, since the fits may be due to idiopathic epilepsy, and the two diseases only accidentally coincident, or indirectly connected. But if the convulsions begin locally, and thus indicate some local change in the brain, a history of constitutional syphilis makes it extremely probable that there is organic disease of syphilitic nature. In the same manner a history of syphilis gives additional weight to other symptoms of organic disease, whatever may be their character.

Convulsions may not only result from active brain disease, but also from brain disease which is no longer in an active state, which has become quiescent, or has even retrogressed, so that merely a cicatrix may be found on post-mortem examination. Such disease may be one which, in its active state, commonly causes convulsions, such as a cerebral tumour. The other symptoms, as headache, optic neuritis, paralysis, may pass away, but the convulsions persist by a continued morbid action of grey matter which was damaged by the irritation of the lesion in its active stage. For instance, a man, who had constitutional syphilis, suffered from convulsions, optic neuritis, and severe headache, and a syphilitic tumour of the brain was diagnosed. The optic neuritis and headache passed away, but the fits persisted, and some time afterwards he died in a series of severe convulsions, killed apparently by their violence. Post mortem there was found the atrophied remains of a gumma in the under part of the right frontal lobe. In a large number of these cases the convulsions begin locally, and are thus of the character to suggest organic disease. Whether they begin thus or not, the history of the case shows that at the onset there were symptoms of organic disease, and usually makes the nature of the case clear. Frequently also some of the early symptoms persist, as hemiplegic weakness, optic-nerve atrophy such as is produced by neuritis, hemiopia, &c.

In a very important group of cases an acute lesion of the brain, in most if not in all cases, softening from thrombosis or embolism, causes at first hemiplegia and afterwards convulsions, which may persist as long as the individual lives, although the hemiplegia passes away wholly or in part. These are the cases of 'post-hemiplegic epilepsy,' described at length in Chapter V. In many cases the disease dates from childhood, but in most the history of the onset will indicate the nature of the case. Whenever epilepsy begins with severe convulsions in early life, especially if these occurred during the course of, or after, an acute specific disease, enquiry should be made for hemiplegia after the early fits. The parents frequently omit to mention it, attaching chief importance to the convulsions. In most cases the recurring convulsions affect first or only the side on which the hemiplegia existed, and frequently begin locally.

*General Paralysis of the Insane.*—In some cases of general paralysis, recurring convulsions, often unilateral, and sometimes varying in seat, constitute the first symptom of the disease, and the affection may thus in the early stage be mistaken for epilepsy. In most cases other symptoms of the disease are present, tremor of lips or tongue, defective articulation, or a tendency to optimism. If these symptoms are detected in a case of short duration, in an adult man, the disease is probably general paralysis and not epilepsy.

*Simulated Convulsion.*—Epileptic fits may be simulated, and are chiefly shammed by professional beggars [1] and by soldiers. Commonly the pretended attack is so unlike a true epileptic fit that its nature is at once recognised. Exact simulation is only possible by a person thoroughly familiar with the characters of the disease. Even then the initial pallor and dilatation of the pupils cannot be produced, although it is said that the insensibility of the conjunctiva may be. The artificial convulsion may bear some resemblance to the aberrant forms of genuine epileptic fit with extensor rigidity of limbs and fine tremor-like clonic spasm; but the absence of the pupillary symptoms will usually enable the nature of the case to be recognised, and circumstantial evidence, the occurrence or repetition of attacks under conditions in which they may be advantageous to the individual, often first awakes suspicion. It is necessary, however, to be aware of the characters of the aberrant forms of genuine seizures, lest the grave injustice be done of treating a real sufferer as a malingerer, a danger which is probably greater than its converse.

To sum up. In any case of convulsions the first step in diagnosis must be to ascertain whether the convulsion is epileptic or hysteroid, and, if there is hysteroid convulsion, whether this occurs alone or succeeds an epileptic fit. Having ascertained the occurrence of epileptoid attacks, if these are recent, any indications of organic brain disease or sources of reflex irritation must be searched for and excluded. The former should be especially thought of if the fits begin locally. The possibility of the attacks being due to alcoholism, renal disease, and lead poisoning must also be borne in mind, if the patient is an adult. The circumstances under which the first fit occurred should then be ascertained, and search made for a history of past hemiplegia or the indications of persistent weakness. The mental condition of the patient must be ascertained to enable commencing general paralysis to be excluded. If none of these conditions can be found, the fits are probably due to idiopathic epilepsy, and the probability of this is increased if any history can be obtained of inherited tendency, or that convulsions or attacks of *petit mal* have occurred in earlier life. It should be remembered that in the middle and upper classes the knowledge of such inherited tendency or earlier attacks is often carefully concealed from the sufferer himself.

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[1] The simulation of epilepsy is a matter of careful training among professional beggars and criminals, to whom it is useful, not only as a means of obtaining alms, but also for escaping penal labour. An interesting account of the confessions of one of these individuals, who succeeded for several years in deceiving English police and prison surgeons, and was ultimately detected in America, has been given by Dr. C. F. Macdonald in the *American Journal of Insanity* for July 1880.

## CHAPTER XII.

### *PROGNOSIS.*

THE prognosis in epilepsy involves several separate questions: (1) The danger to life; (2) The prospect of a spontaneous termination of the disease; (3) The prospect that by treatment the disease may be (*a*) cured, or (*b*) the attacks arrested.

(1) *The danger to life* in epilepsy is not great. Alarming as is the aspect of a severe epileptic fit—imminent as the danger to life appears when the patient is lying senseless, with livid, swollen and distorted features, and convulsions which almost asphyxiate him, looking 'as if strangled by the bow of an invisible executioner,'[1] it is extremely rare for a patient to die during a fit. The chief danger of death in an attack is the liability to accidental asphyxia, in consequence of the occurrence of an attack during a meal, when food may get into the air-passages, or of vomiting after an attack with the same result, or in consequence of the patient, in bed, after an attack, turning on to the face and being suffocated in the post-epileptic insensibility. It is for this reason that the danger to life is much greater in the cases in which there exists this tendency to turn on to the face than in others.

There is also some risk of death by other forms of accident to which the attacks expose the patient. Few epileptics of the lower class, whose attacks occur without warning, escape an occasional burn from falling on to the fire, and the burns thus received are sometimes serious and dangerous. But the commonest mode of accidental death in epilepsy is by drowning. The fit not only occasions the fall into the water, but effectually prevents any effort to escape, and often interferes with any attempt at rescue. Hence epileptics are sometimes drowned in a very small depth of water, as in a ditch.

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[1] Radcliffe.

The danger of such accidental death is unquestionably greater than that of death from the direct severity of a fit. The latter is excessively rare, especially when the frequency of severe fits is taken into consideration. Very rarely, however, epileptics pass into what is termed the 'status epilepticus' (described at p. 193), in which severe attacks recur very frequently, recovery from one being imperfect before another comes on. This state is one of considerable danger; it is, however, so rare, and the liability to it is so small, that it cannot be regarded as measurably increasing the risk of death in consequence of the disease.

(2) *What is the prospect of a spontaneous cessation of the fits?*—The tendency of the disease is to self-perpetuation; each attack facilitates the occurrence of another, by increasing the instability of the nerve elements. Hence the spontaneous cessation of the disease is an event too rare to be reasonably anticipated in any given case. Occasionally, however, convulsive attacks in infancy, which continue after all cause to which they could be attributed has passed away, cease spontaneously at four or five years of age; sometimes they do not afterwards recur, but too often they come on again at the period of the second dentition or at puberty. Attacks which have commenced before, and continued until puberty, rarely cease at that period. The expectation, firmly rooted in the popular mind, that attacks before the establishment of menstruation will cease when this has occurred, is unjustified by facts. My own observations on this point agree perfectly with the statements of Herpin. It is extremely rare for menstruation to make any difference in such cases. After twenty years of age, spontaneous cessation does sometimes occur, and it becomes more frequent as life advances. It is, I believe, a more frequent

event than writers on the disease have usually admitted, but is not sufficiently common to be reckoned on as an element in prognosis.

Marriage, as a rule, makes no difference to epileptics. The attacks occur afterwards with the same frequency and severity as before. In some patients they cease during pregnancy, but usually recur when the pregnancy is over.

(3) *The probability of cure or arrest by treatment.*—The probability of a spontaneous arrest of the disease being so small, the next question becomes of paramount importance: What is the prospect of cure of the disease, or of arrest of the fits by treatment? The facts relating to the 'cure' of the disease are extremely meagre, since very few cases can be watched sufficiently long to enable a cure to be confidently affirmed. As far as is known, however, the only method of cure is by obtaining arrest of the fits for a considerable time, and hence the answer to the second part of the question becomes practically the answer to the whole, as far as it can be given.

What is the prospect, in any given case, that an arrest of the fits can be obtained by treatment? The indications of the prognosis have been materially changed by the introduction of the bromides as remedies for epilepsy. Not only do they arrest fits far more frequently than any other remedy, but they are effective in many cases which, according to experience previous to the introduction of these remedies, would have been regarded as most unpromising. Hence, by their use, the conditions of the prognosis have been essentially changed. In order to ascertain how far the various conditions of the disease can be taken as indications of the amenability of a case to treatment, I have compared the facts of 43 cases, in which no good was done by any method of treatment, with those of 100 cases in which the attacks were arrested by treatment as long as the patients remained under observation, all cases known to have relapsed being excluded. The number of cases is not large, but the conclusions agree with those which have been impressed on me by the observation of individual cases.

The points examined with reference to prognosis are (1) sex; (2) age at commencement; (3) duration; (4) inherited tendency; (5) exciting cause; (6) frequency of attack; (7) condition of patient under which attacks occur; (8) existence of an aura; (9) mental state; (10) character of the fits.

*Sex.*—In the cases which were unimproved the females preponderated (as in the whole of the cases of epilepsy analysed, see p. 7). In the cases in which the attacks ceased under treatment, the sexes were affected equally. Hence the males were more numerous, and the females less numerous, among these cases, than among the entire series, and it may therefore be concluded that the prognosis is slightly better in males than in females.

*Age.*—The following table shows that age has a distinct influence on prognosis. The percentage of the unimproved cases to the whole is 30 (43 : 143 :: 30 : 100). The percentage of the cases commencing at each age, arrested and unimproved, is stated; and between brackets is indicated the excess of the arrested or unimproved cases, at each period of life, over the proportion for the whole, 30 and 70 per cent. respectively.

| | Cases | | Percentage | |
|---|---|---|---|---|
| | Unimproved | Arrested | Unimproved | Arrested |
| Under 10 . . . | 14 | 29 | 32·5 (+2·5) | 67·5 |
| 10–19 . . . . | 23 | 45 | 34 (+4) | 66 |
| 20 and over . . . | 6 | 26 | 19 | 81 (+11) |
| | 43 | 100 | 30 | 70 |

Thus the proportion of the cases commencing under 20 in which arrest was obtained is considerably less than the proportion of cases commencing over 20, the difference amounting to about 13 per cent. The period of the first twenty years of life at which the disease commences has little influence, but the prognosis is a little better in the cases which commence under ten than in those which commence between 10 and 20; arrest is more frequently obtained. The cases which commence in women at the second climacteric are also obstinate, although not sufficiently numerous to be separately given.

*Duration.*—The influence of the duration of the disease, examined in the same way, is shown in the following table:—

| | Cases | | Percentage | |
|---|---|---|---|---|
| Duration | Unimproved | Arrested | Unimproved | Arrested |
| Less than 1 year | 4 | 19 | 17 | 83 (+13) |
| 1–4 years | 14 | 37 | 27 | 73 (+3) |
| 5–9 „ | 9 | 20 | 31 (+1) | 69 |
| 10 years and over | 16 | 24 | 40 (+10) | 60 |
| | 43 | 100 | 30 | 70 |

The result is in agreement with the conclusions of all writers on the subject, that the prognosis is favourable in inverse proportion to the duration of the disease. The percentage of cases arrested declines gradually from 83, in the cases which have lasted less than a year (which is 13 per cent. above the average), to 60 per cent. in the cases which have lasted ten years and more, which is 10 per cent. below the due proportion. The prognosis is therefore by far the most favourable in cases which have lasted less than a year. Having regard only to the probabilities that treatment will arrest the fits, or will have no effect whatever upon them, it appears that if the disease has lasted less than five years the probability is in favour of arrest, but that if the disease has existed for more than five years it is more likely that no effect will be produced than that complete arrest will be obtained. Of course, at any age the probability of amelioration, of a diminution of the fits, it may be to one-fiftieth or one-hundredth of their former number, is always greater than either of the two eventualities which are now, for the sake of bringing out more clearly the prognostic indications, alone under consideration.

*Hereditary predisposition* might reasonably be expected to have an unfavourable influence on prognosis, to render it less likely that an arrest of the fits would be obtained. As Herpin first pointed out,[1] such is not the case. From observation of individual cases I have been strongly impressed by the frequency with which heredity exists in the cases in which treatment has the most marked effect. The fact is clearly brought out by a comparison of cases. Of 33 in which no improvement was obtained from treatment, heredity existed in ten only, or 30 per cent. Of 100 cases in which the fits were arrested by treatment, heredity existed in forty-five, or 15 per cent. more than in the other series. Thus heredity seems to render it easier to obtain a cessation of the fits. The explanation may be that a slighter exciting influence is effective in such predisposed cases, and is more easily counteracted. It does not of course follow that a permanent cure is more readily effected in such cases. The greater readiness with which the fits are arrested may be counterbalanced by a greater tendency to relapse. A similar fact has been observed with regard to insanity; heredity does not lessen the probability of recovery from a given attack.

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[1] *Loc. cit.* p. 515.

Illustrations of this fact are often met with, such as was strikingly presented by the following case:—

A young man, whose mother's two sisters and one brother were epileptic, had his first fit at nineteen, and soon afterwards came under treatment. The attacks were at once arrested, and they remained absent for two years. Then, at twenty-one, they recurred, and, having had two fits in one week, he came back to the hospital. Each fit was preceded for an hour or two by a 'nasty feeling of numbness in the tongue,' but there was no immediate warning. In the fit consciousness was lost, and his tongue bitten. A scruple of bromide twice a day was ordered, and he had no other fit, and after two years' treatment he was discharged. The fits remained absent for a year, and then he had some more, and again came to the hospital. The attacks at once ceased under the same treatment, but he had occasional 'sensations' in the tongue, which became more frequent when the same dose of bromide of sodium was substituted for bromide of potassium, but on the resumption of the latter with Indian hemp, although only once a day, the sensations ceased, and he has now (April, 1881) had no symptom for a year.

An equally striking illustration was presented by the patient, mentioned on p. 11, fourteen of whose relations suffered from epilepsy. The attacks commenced at ten years of age, and she was thirty-seven when she came under treatment. The interval, before the treatment commenced, was two weeks. The attacks occurred during sleep only, and never during pregnancy. They were severe epileptic fits, with tongue biting. Under treatment the attacks practically ceased. As long as she was taking ℈j or ℥ss of bromide at bed-time she had no fit, but after she had been a year, eighteen months, or two years without an attack, she would discontinue the bromide and presently have another fit. This has now gone on for seven years, and the bromide is just as effectual as when she first came under treatment.

Several other illustrations of the same fact will be mentioned in the section on 'Treatment.'

*Exciting Cause.* —The recognition of an exciting cause for the disease, *i.e.* a cause for the first fit, has little influence on the prognosis. Such a cause can be traced in about the same proportion of the cases which are arrested, and of those which are not influenced by treatment. No indication can be drawn from the character of the exciting cause in the cases in which one can be traced.

*Frequency of Attack.*—The frequency with which attacks occur before treatment is commenced affords important information regarding the effect which may be looked for, as the following table of 100 cases shows:—

| | Cases | | Percentage | |
|---|---|---|---|---|
| | Unimproved | Arrested | Unimproved | Arrested |
| Attacks daily . . . | 7 | 1 | 18 | 1·8 |
| 1 day to 1 week . . | 11 | 29 | 29 | 46·7 |
| 8 days to 1 month . . | 16 | 15 | 42 | 24·2 |
| Over 1 month . . . | 4 | 17 | 11 | 27·3 |
| | 38 | 62 | 100 | 100·0 |

Thus the prospect of a complete arrest of the fits is extremely small if severe attacks occur daily. It is greatest if the attacks occur at a longer interval than one month. There is not, however, a simple relation between length of interval and prognosis, because attacks which occur at the regular interval of a month are less frequently arrested than those which occur at a shorter interval.

*The condition in which the attacks occur*, whether during the waking or sleeping state, or both, is also of significance. I find this point noted unfortunately in only 19 of the cases in which no improvement was effected, but the figures, as far as they go, are very suggestive. Attacks occurred only during sleep in 4 cases, only during waking in 4 cases, and in 11 during both states. That is, they occurred in both states in a greater number of cases than in one only, the proportion being nearly 3 to 2. On the other hand, in 100 cases in which arrest was effected, the fits occurred only in the waking state in 45 cases, only during sleep in 31 cases, and during both waking and sleeping in only 24 cases. Thus they occurred in only one state in three times as many cases as in both sleeping and waking. It is clear, therefore, as far as these figures go, that the prognosis is far better if the attacks occur in one state only, than if they occur in both.

*Aura.*—The occurrence of a distinct aura renders the prognosis a little more favourable. Of the unimproved cases, an aura was present in 13 and absent in 28; it was thus absent more than twice as frequently as it was present. In the arrested cases an aura was absent in 53, and present in 49: it was thus present and absent with nearly the same frequency. The form of aura seems to have little influence on prognosis, with the exception of the cases in which the warning consists of vertigo, or of a visual sensation, or of a unilateral peripheral aura, all of which render the prognosis slightly more favourable.

*Mental Changes.*—The existence of considerable mental change renders the prognosis unfavourable. Mere slight loss of memory is, however, of little significance. It may be recovered if the attacks are checked by treatment.

*Character of Fits.*—Regarding the influence on prognosis of the character of the fit, it is to be remarked that the major attacks are influenced by treatment much more readily than the minor fits. The former often cease before the latter are materially lessened, and it is not infrequent for the minor attacks to persist in spite of treatment, although the former are completely arrested.

To sum up the prognostic indications in idiopathic epilepsy. The prognosis is slightly more favourable if the patient is of the female sex, and distinctly more favourable if the disease begins over twenty than if it commences between ten and twenty. It is better the shorter the duration of the disease, and when the disease is inherited than when no heredity can be traced. It is better the longer the interval between the severe fits, and is least so in the cases in which attacks occur daily. It is better if the attacks occur in the sleeping or in the waking state only than if they occur in both. It is better if there is no considerable mental change, and if the attacks are all of the severe variety. than if there are minor seizures, and better if the attacks are preceded by an aura than if they occur without warning.

*Post-hemiplegic Epilepsy.*—In the cases in which the attacks are 'post-hemiplegic' the prognosis is much less favourable than in the idiopathic cases. It is influenced by duration of the disease and interval between the attacks, just as in the idiopathic form, but, as might be expected, the effect of remedies is less marked, and these cases furnish some of the most obstinate cases which come under treatment.

*Hysteroid Attacks.*—The prognosis in hysteroid attacks is on the whole more favourable than in the purely epileptic seizures. It is, however, of great importance to ascertain whether the hysteroid attacks occur alone, or are consecutive to minor epileptic fits. The latter may persist even though the former are arrested. In pure hysteroid fits the prognosis depends partly upon the existence of other pronounced manifestations of hysteria, which, as indicating a profound affection of the brain, renders the immediate prognosis less favourable, and partly on the degree to which the patient can be removed from unhealthy home influences. Patients often remain entirely free from fits during their residence in a hospital, and relapse as soon as they return to some annoying and disturbing conditions at home. The prognosis is rather more favourable if the attacks are severe than if they are slight, but are least so in the cases in which the fits are of the mixed or intermediate character between hysteria and epilepsy described on p. 176, in which treatment, whether directed against the hysteria or the epilepsy, is often ineffective.

The ultimate prognosis in cases of pure hysteroid convulsion is almost always favourable. The attacks after a time usually cease.

# CHAPTER XIII.

## *TREATMENT.*

THE treatment of epilepsy is a subject on which numerical analysis gives little help. A large number of cases are under observation too short a time to enable the effect of remedies to be fairly estimated, and, of the cases in which benefit is derived, we have no means of ascertaining how many relapse when treatment is discontinued. Of the cases analysed, the effect of treatment was noted in 562. Complete arrest, for a time, was effected in 241; in most of these the fits remained absent as long as the patients were under observation, and some were heard of after the treatment was discontinued, and were still free from fits. In 266 cases improvement short of arrest was obtained, the fits being reduced, in many, to one-thirtieth, one-fiftieth, and even one-hundredth of their former frequency. In fifty-five cases no improvement was effected by any method of treatment.

We have still too little definite knowledge of the intimate pathology of epilepsy to permit safe generalisation regarding the possible mode of action of the several drugs which are found, clinically, to be useful. All that can be done is to recognise the kind of action which they have been found, by experiment, to exert upon the nervous system.

The character of the cases in which this or that drug is especially useful, *i.e.* the 'indications' for special treatment, is a subject of the greatest practical importance. There is no point in therapeutics, however, more open to fallacy, or on which more generalisations have been published which subsequent observation has proved to be inaccurate. While pointing out, therefore, when possible, the class of cases in which this or that method of treatment has seemed most useful, I have thought it better to illustrate the effect of remedies by the brief narration of some cases in which that effect was well marked, rather than to formulate a series of precise 'indications' which experience might fail to confirm. The cases described are selected from a larger number, as those which seemed most instructive.

*Bromides.*—Since the introduction of bromide of potassium as a remedy for epilepsy, it has almost superseded other drugs in the treatment for the disease. The signal benefit which, in the majority of cases, attends its use, has rendered the administration of bromide and the treatment of epilepsy almost equivalent expressions. But it is most important that attention should not be fixed upon bromide too exclusively. In the majority of cases its influence is far greater than that exerted by any other known agent; in an important minority of cases, however, bromide fails to do good, and in many of these other drugs are effective. Moreover, the influence of bromide is, in the majority of cases, transient, and not permanent. The effect ceases when the administration is discontinued. This is often urged as an objection to its use. It is certainly desirable to continue the search for remedies which shall have a more permanent effect, but it may be doubted whether the want of permanence in the action of bromide is to be laid at its door. It is perhaps the result of the nature of the disease, for the same fact is true of all the remedies which have been hitherto employed; the same complaint was repeatedly made regarding other remedies in the pre-bromidic days. In most cases their influence lasts little longer than their administration, and it is probable that bromide can show more permanent cures than all the other remedies put together, and that it can show ten times as many instances of amelioration which fall short of cure.

Of the three alkaline salts of bromide, that of potassium deserves, I think, as it has popularly received, the first place. It is certainly superior to the bromide of sodium. I have substituted the sodic for the potassic salt in fifty cases and noted the effect. In a good many no difference was perceptible, but in at least half the cases the sodic salt was distinctly less useful, dose for dose. Bromide of ammonium is in most cases as effective as bromide of potassium, sometimes apparently more powerful, and it seems to have even less than the very slight depressing influence now and then produced by bromide of potassium. The relative power of the bromides, according to my experience, is not proportional to the quantity of bromine which they contain. The amount in each of the bromides is as follows:

| | |
|---|---|
| Bromide of ammonium . . . . . | 81 per cent. |
| „ potassium . . . . . | 67 „ |
| „ sodium . . . . . . | 77 „ |

Bromide of lithium has been recommended by Weir Mitchell, and, were the influence of these salts proportional to the amount of bromine they contain, it might be expected to be of special use, since it contains no less than 92 per cent. I have watched its effects in a considerable number of cases, but have not been able to trace any superiority in its action.

Bromides are said to cause contraction of the small vessels of the brain;[1] but it is exceedingly doubtful whether any of their influence in epilepsy is due to this action. They have unquestionably a direct effect on the nerve elements, diminishing, for instance, reflex action in the spinal cord, and it is probable that the mental dulness and somnolence produced by large doses are also due to a direct action on the nerve-cells of the brain. If we believe that the primary change in epilepsy is a morbid action of the nerve-cells, it is reasonable to conclude that the drug does good in epilepsy by directly influencing them. Agents which increase reflex action, such as strychnia, are now believed to do so by lessening the resistance in the nerve-centres involved. Bromide diminishes reflex action, antagonises strychnia,[1] and it is probable that it does so by increasing the resistance in the nerve-centres. If the view suggested in the preceding pages is correct, that the morbid state in epilepsy is essentially an instability of the resistance in the cells, it seems probable that the influence of bromide in the disease is produced by increasing the stability of that resistance.

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[1] By Solkowski. The observations, which were made on trephined animals, stand, according to Nothnagel, 'very much in need of confirmation.'

The common method of the administration of bromide in epilepsy is by what may be called the *method of minimum doses*, that is, the continuous administration of the smallest dose which will suffice to arrest the fits, and only giving a large dose when a smaller quantity fails to effect this object. Given thus, it needs to be given frequently, since the smaller the dose the briefer is its influence. It is, however, often important, in order to secure regularity in taking the medicine, not to give it more frequently than is absolutely necessary. When the attacks always occur at a certain time, a single daily dose will usually suffice. If possible, this should be given not more than two or three hours before an attack is expected. This is contrary to some opinions which have been expressed, but I have several times known attacks to be arrested by bromide given an hour or two before the time for their occurrence, which were not arrested when the drug was given twelve hours earlier. If the attacks occur at various times, it is commonly necessary to give the bromide twice or three times a day, or else to give larger doses, the action of which is of longer duration. I have several times observed that a daily dose of a drachm, given in two (half-drachm) doses, did not arrest the fits, although they ceased if it was given in three (scruple) doses.

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[1] According to Schroff, the tetanus of strychnine-poisoning can be arrested by bromide.

When bromide is thus given continuously, the daily quantity may vary between fifteen grains and two drachms, according to the age of the patient and the effect of the drug. Few patients are able to bear more than a drachm and a half a day, for long, without becoming bromised —lethargic and dull, physically and mentally, with cold extremities and feeble pulse. The best results are usually obtained with a dose not exceeding one drachm daily. If this does not succeed in arresting the fits, they are rarely arrested by larger doses, and combinations of bromide with other drugs offer greater promise of relief.

The effect of bromide is in many cases cumulative. The attacks are not at once arrested, but they become less frequent and cease after the medicine has been continued for two or three months.

The treatment of epilepsy by bromide or other drugs is for the most part, as already stated, a question of arresting the fits, or, failing this, of producing the greatest possible diminution in their frequency and severity. The arrest of fits is of course not equivalent to the cure of the disease. In only too many cases, when the attacks have been arrested for a long time by treatment, they recur when that treatment is discontinued. In some cases, however, the patient remains free from fits for one, three, or five years, and then the attacks recur. In the majority of cases which relapse, recurrence takes place within a few months of the cessation of treatment, and if, after such cessation, the patient has passed a year without an attack, there is good ground for the hope that they will not return, an expectation which is strengthened by every subsequent year of freedom. If a relapse occurs, the fits are somewhat less readily arrested by the same treatment than in the first instance. This constitutes a great difficulty in the management of these cases. A very frequent occurrence is, that a patient, after a few months' freedom from fits, discontinues the medicine and relapses. He comes again; the fits are again stopped, but less readily than before; again he omits the treatment and the attacks recur, and are then not completely arrested. Hence it is most important to maintain by long treatment the initial arrest of the fits. The longer the period since an attack, the greater, *cæteris paribus*, is the probability that the arrest will continue after the medicine is discontinued. Hence treatment should be continued, without any reduction in dose, for six or twelve months after the last fit, the period varying according to the duration of the disease and frequency of attacks before treatment was commenced, and the readiness with which cessation is obtained. The dose should then be gradually reduced, and, if possible, it should not be entirely discontinued until another equal period has elapsed. It should never be suddenly left off. A good plan, for instance, is as follows: If the attacks cease on a scruple of bromide three times a day, this should be continued for twelve months; given twice a day, for three months more; then half a drachm once a day substituted, and reduced three months later to a scruple, and three months later to ten grains, which may be omitted at the end of the second year.

At any period 'threatenings' of an attack, *e.g.* a 'sensation,' such as that which preceded the attacks, should lead to an immediate increase in the dose. But it must be remembered that patients, dreading a recurrence, are very apt to regard as 'threatening,' symptoms which really have not that significance.

In the following case there is good reason to believe that the patient is cured, although there was an indication of a family tendency to the disease:—

A girl, Sarah W., aged 27, whose brother suffered from fits at nine, had her first attack also at nine, immediately after a fright. She had had them ever since, and when she came under treatment, January 1874, they occurred at intervals of about six weeks. Each was preceded by a feeling of 'lostness' for a few seconds, then her arms became stiff, and she lost consciousness, and the fits were followed by heavy sleep. The sensation sometimes occurred without a fit. A scruple of bromide was given three times a day. She had no other fit. For twelve months she had occasionally a 'faint feeling,' and at the end of that time the bromide was reduced to two doses a day. The 'faint feelings' became less and less frequent, and at the end of the second year the bromide was omitted altogether, and quinine was given for a few months. I have lately (April 1881) heard that this patient has continued perfectly well. It is now five years since the cessation of treatment, and seven years since the last fit, and there has been no indication of recurrence. She married four years ago, and has two healthy children.

In the case of children the dose of bromide should of course be smaller than for an adult. Over six years of age, however, it is desirable to give not less than ten grains. The efficacy of this dose is illustrated by the following cases, of which the first is one of recent epilepsy. In the third case a still smaller dose was efficacious.

Thomas H., aged 10, without neurotic family history, who was brought ten weeks after the commencement of the fits. The attacks were epileptoid and severe, attended with tongue-biting, and five occurred in the week before treatment was commenced. Ten grains of bromide twice a day were ordered, afterwards changed to five grains three times a day. From the first dose of bromide to the present time, now two years, he has not had another fit. He is still under treatment.

Albert H., without indication of heredity, had his first fit at seven months old, without any indication of cause. The attacks occurred at intervals of one to six months, both sleeping and waking, until he came under treatment, at the age of ten, in January 1879. He was ordered ten grains of bromide of potassium every morning. From the first dose of the medicine he never had another fit. In February 1880 the dose was lessened to five grains, and in April he discontinued treatment, and I have lately (April 1881) learned that he has continued perfectly well during the past year.

William C., aged 9. The attacks commenced at eight, without known cause, and occurred once a fortnight when he came under treatment, being preceded by an epigastric aura. He took five grains of bromide twice daily for twelve months, without a single fit, and was then (three years ago) discharged, with directions that he was to be brought to me again if there was any recurrence, but he has not been heard of since.

Bromide is as effective in the disease in very young children as in patients of older age, as the following two cases testify.

A boy, aged 4, began to suffer from fits at six months. The only indication of rickets that could be ascertained was that he only walked at a year and eight months. The fits were frequent and severe. There was no history of worms. Five grains of bromide were given three times a day. No attacks occurred for five months, and then he had several without discoverable cause. The dose of bromide was increased to seven grains, and he had no attack for twelve months, and then, the dose having been previously lessened, he ceased to attend.

The recurrence of fits which have been arrested by treatment may be spontaneous, or it may occur only on a considerable exciting cause. For instance, in the following case an accident reproduced attacks which appeared to have been effectually arrested.

Fanny P., whose father's brother was insane, had her first fit at ten, while unconscious after a fall out of a 'boat-swing.' She came under treatment at the age of fourteen, having then a fit every fortnight. Under treatment (which was continued for three years) the attacks ceased entirely. She then took no medicine, and had no attack for five years. At the end of that time she was thrown out of a dogcart, and again struck her head; she was rendered insensible, and had a fit a few minutes after the accident. They recurred subsequently every ten days, until she came under treatment nine months later, when they again ceased under bromide.

If a relapse occurs after bromide treatment has been discontinued, the drug should be continued the second time for a much longer period than the first, as in the following case, which illustrates also a relapse after a long period of freedom.

George R., whose father's mother was insane, had his first fit, without known cause, at seventeen, and came under treatment at the age of twenty-two. Bromide (℈j bis) at once arrested the fits, and after he had been under treatment for nine months, having no fit, he ceased attendance, and remained free from fits for four years. He then had another attack after running quickly up to the top of a factory. Bromide was again given, and, the treatment being continued, he has now passed a year and three quarters without an attack.

When bromide 'cures' epilepsy, removing altogether the state of the nervous system to which the attacks are due, this effect must be ascribed to a permanent nutritive change of the same character as that functional change which is produced by its presence in the system. To express its effect in the language of the hypothesis already advanced, it may be said that the presence of bromide increases, functionally, the stability of the resistance of the nerve-cells. The effect may cease when the bromide ceases to be present. But if this functional effect is maintained for a long time, nutritive changes occur by which the effect is maintained after the bromide is withdrawn. In cases in which there is reason to hope that this result may be obtained—that is, in cases in which the attacks are readily arrested by bromide—it has seemed to me desirable to endeavour to aid in the production of this nutritive change by subjecting the patient to the full influence of bromide *for a short time*, by giving doses much larger than are necessary to arrest the attacks, giving in fact, for a few weeks, the largest doses the patient will bear. This may be termed the method of *maximum dose treatment*. It has seemed to me to be of distinct value. The object is to give the nervous system, as it were, a series of blows with bromide in order to facilitate the occurrence of the condition which bromide produces in patients who are cured of epilepsy by its use. The method I usually adopt is to begin with doses of two or three drachms of bromide every second or third morning, and increase the dose to four drachms every fourth morning, and six drachms or an ounce every fifth morning. These large doses should be given after breakfast, in a tumberful of water. If the salt is not well diluted, they cause epigastric pain and vomiting. As a rule, when the dose reaches half an ounce, some drowsiness and mental dulness follow during the rest of the day, but are gone the following day. Some patients complain only of headache, a dull constricting sensation, which is relieved by the application of cold. I do not increase the dose beyond that which produces transient lethargy and mental dulness. The susceptibility of different patients to these large doses varies exceedingly. Some patients cannot bear more than four drachms; in others a dose of an ounce produces no change from their normal state. I have not found that a larger dose than an ounce is borne, ten or twelve drachms being usually vomited. These doses usually lower the frequency of the pulse 8–14 beats, but make no appreciable difference to the temperature or to the reflex actions.

The maximum dose should be reached in two or three weeks, and repeated three or four times, and the doses then gradually reduced, so that the whole course lasts six or seven weeks. It is to be remembered that the object of this method is to attempt to further the *cure* of the disease. It is therefore only suitable for cases in which the attacks are influenced in a marked degree by bromide. Unless the attacks cease entirely when a dose of four drachms is reached, the prospect of curing the patient is too small to make it advisable to persevere with the treatment. If the bromide is altogether omitted at the end of the six or seven weeks' course, the patient as a rule remains free from fits for four or six months, a much longer freedom than follows, in similar cases, so short a period of treatment by bromide in ordinary doses. The attacks do, however, recur in most cases if all treatment is relinquished, and hence bromide should not be altogether omitted on the cessation of the course, but should be continued in ordinary doses. At the end of the course, if it is thought desirable, the patient may be left without bromide for a week or two before it is resumed. The method is thus intended as an initial step in the treatment, with the object of rendering more permanent the effect of the subsequent treatment. There is considerable reason to believe that it does so, although I have not employed it in this manner long enough to be able to adduce any evidence of permanent effect. Most of the patients in whom I have adopted it have omitted the bromide altogether and have relapsed after four, six, or eight months' freedom. The attacks recurred very frequently in the patients thus treated, and according to ordinary experience they would have relapsed within a week of the end of six weeks' treatment with ordinary doses. Hence the facts, I think, justify the anticipation that this method of an initial course of maximum doses will be found a useful help in the attempt to cure epilepsy by bromide.

The inconveniences of bromide are twofold: first, the class of symptoms termed bromism; and secondly, the bromide rash.

By bromism is meant the physical feebleness and mental dulness which sometimes result from the drug, and which may amount to a semi-imbecile condition, with drawling speech and dribbling mouth. This condition results from continued administration in moderate doses, such as 60, 90, or 120 grains a day. As a continuous state, I have never known it result from the large, infrequent doses just described. Its occurrence is distinctly influenced by individual idiosyncrasy, especially by a weakened nervous system as part of the original disease. It is very rarely seen in persons whose health, physical and mental, is, to begin with, up to the average. On the other hand, it is often most troublesome, and is produced by very small doses, in patients with imperfect brain-power as the effect of the disease or of its cause. It is certain, also, that the effects of the disease on the mental state are very often attributed unjustly to the bromide. Not only is this the case with the symptoms which result from the disease, but occasionally, in rare cases, the cessation of fits, from whatever cause, is attended with a diminution of mental power. Patients sometimes find that they are better and clearer-minded after a severe fit than before it; and in such cases, if the severe fits cease, the mental state may become more feeble. This is especially the case when minor attacks do not cease, or when they replace the severe fits. It is occasionally seen when there are no minor fits. If bromide has been used, such mental deterioration is commonly ascribed to the bromide when it is really due to the change in the disease. For actual bromism there is no remedy except a diminution in the dose, and, in such cases, we have to seek, in combinations of bromide with other drugs, the increased power which we need.

The second inconvenience of bromide is the acne which it so often produces on the skin. Its occurrence is, like bromism, very much a matter of individual idiosyncrasy. Many patients can take large doses of bromide daily for years without a trace of eruption, while in others a single drachm is sufficient to produce a crop of spots. The facility with which the rash occurs with the different bromides seems to depend upon the amount of bromine they contain. Between bromide of potassium and sodium there is little difference; but I have known the rash to appear at once when bromide of ammonium was substituted for bromide of potassium, although the latter had been taken for a considerable time without an effect on the skin. The amount of eruption may often be observed to vary with the dose of bromide.

The common form of the rash is, as is well known, pustular, the red swelling being commonly large and the point of suppuration small. Almost as frequent, however, and more so at the commencement of the rash, are small pustules, with little redness, and papules which do not always reach the stage of pustules, and are often hard. I have known this form of rash to be mistaken for varioloid. It has been said that the white centres of the bromide pustules do not contain true pus, but only caseous material. This is often the case, but sometimes they contain true pus. Large pustules are occasionally seen in which there are many minute white points of cheesy or purulent material, and occasionally there are large and superficial bleb-like collections of pus surrounded by a little redness. Guttmann has shown that the presence of bromine may readily be demonstrated in the contents of the pustules. The larger pustules are painful and leave permanent scars, so that, if numerous, they may cause considerable disfigurement, from the pitting and from the reddening and thickening of the skin between the pustules.

This troublesome symptom may be almost entirely prevented or removed by the addition of arsenic to the bromide, an obvious expedient, which was probably employed independently by more than one physician. It was recommended by Echeverria in 1870.[1] From three to five minims of liq. arsenicalis added to each dose of bromide will usually remove the rash, if moderate, and greatly diminish it, if considerable. When the eruption has thus been removed, if the arsenic is omitted, the bromide being continued, the eruption returns.

The following cases illustrate the effect:—

S. S., a man, aged 38, had taken bromide for five years, and during the whole of that time he had had a large amount of acne upon the face. In the summer of 1877, while taking a scruple three times a day, his face was covered with coalescing pustules, and presented a most repulsive appearance. The eruption was also abundant on the chest. On reducing the dose of bromide to one half, the acne lessened considerably, but the fits became more frequent, and an increase in the bromide was attended by a corresponding increase in the amount of eruption, which was soon as bad as ever. On September 28 five minims of liq. arsenicalis were added, twice daily. In a fortnight all the spots of acne had disappeared from the face, and those on the chest were subsiding. The arsenic was continued for some months and then discontinued. A month afterwards the face was covered by a fresh bromide rash, and spots of acne had appeared on the back of the neck, on the chest, and on the arms. The eruption reappeared a week after the discontinuance of the arsenic, and disappeared soon after this was resumed.

Louisa S., aged 26, suffering from long-standing epilepsy, had for years been disfigured by long-standing bromide rash. No treatment had availed to lessen the eruption except a discontinuance of the bromide, and the rash recurred as soon as the bromide was resumed. The addition of two minims of liq. arsenicalis to each dose removed every trace of the rash (except a few scars) in six weeks.

Now and then so small a dose of arsenic as was successful in the latter case is insufficient, while a larger dose is effective. Of this the following case is an example:—

A young man, aged 28, who had had fits for sixteen years, and had taken bromide for several years, presented, on March 21, many spots of acne on the face. He was then taking twenty grains of bromide of ammonium three times a day. Three drops of liq. arsenicalis were added to each dose. In a fortnight the face was quite free from acne, but a week later several fresh spots appeared, although the arsenic had been continued. The dose was then raised to five drops, and in a month the rash had almost disappeared.

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[1] *Loc. cit.*, p. 318.

The above cases are sufficient to illustrate the effect. I have described a larger number of cases of the same character in the 'Lancet' for June 15, 1878.

The combinations of bromide with other drugs are of much value in the treatment of epilepsy. In many cases a greater effect is produced by the combination than by either drug given alone. In a considerable number of patients the effect of the several combinations was carefully tested by giving, first, bromide alone for several months, and, when its influence was ascertained, the second drug was added to the same dose of bromide. I will mention several cases in which this method was pursued, and in which the beneficial results of the addition were most clear. In some other cases this method was not pursued, the combination being given from the first, and these cases are not, therefore, so strictly to the point, although they deserve mention on account of the satisfactory therapeutical results.

*Digitalis.*—A very old remedy for epilepsy, it was recommended by Parkinson in 1640, and has been, perhaps for as long a time, a popular remedy for the disease in the west of England. Given alone, I have not found its power to be considerable. It often lessens slightly the frequency of attacks, and sometimes even arrests them for a short time; but I have not met with any case in which, under its use, the attacks remained absent for more than a month or two. It is, however, a most useful adjunct to bromide. In many cases (as in some which will be mentioned) attacks, which continued on bromide only, ceased entirely on bromide and digitalis.

Among the cases which are most strikingly benefited by the combination are those which are complicated by cardiac disease, actual valvular disease, dilatation, insufficient hypertrophy, irregularity, and undue frequency.

The use of digitalis in such cases is an obvious measure. The only cardiac condition in which I have known it to have a prejudicial effect on the attacks is in aortic regurgitation accompanied by considerable hypertrophy, a condition in which the use of the drug is to a large extent contraindicated on cardiac grounds. It is also of considerable use in cases of nocturnal epilepsy. Todd thought that nocturnal epilepsy was especially connected with cardiac disease, but in many patients with heart disease the attacks occur only by day. The combination is also useful in some cases of diurnal epilepsy in which there is no evidence of cardiac derangement, functional or organic.

By what influence digitalis is useful in epilepsy is at present unknown. It steadies the circulation and increases the tone of the small arteries, and thus, doubtless, renders equable the supply of blood to the brain. But its effect on the heart and arteries is produced through the nervous system; that on the heart is supposed to be exerted through the vagus—an indication probably of a central action. Moreover, large doses of digitalis, if continued for some time, cause symptoms of cerebral and spinal disturbance, giddiness, amblyopia, dilatation of the pupil, and even diminished reflex excitability in the spinal cord. These are commonly explained as secondary to the disturbance of the circulation, and the accumulation of carbonic acid in the blood, but it is possible that they are in part due to a direct influence upon the nerve centres.

The greater effect of the combination of bromide and digitalis than of either given alone is clearly shown by the following case:—

In a girl, aged 20, without heredity, one to three fits had occurred weekly since sixteen years of age, distinctly epileptic, with tongue-biting and without warning. Her pulse was 96, irregular in force but not in frequency; the cardiac impulse slapping, but in the normal situation. On tinct. digitalis ♏v ter die, she had one fit a fortnight; on pot. brom. gr. xx bis die, she had a fit each week; on a combination of the two twice daily, she had not a single fit for three months, and then ceased attendance.

The following cases also illustrate the beneficial influence of the combination:—

A girl, aged 21, had two fits at six months old, and no others till thirteen, when they recommenced and continued once a month, chiefly at catamenial periods. They were severe, epileptic in character, occurring both when awake and when asleep, and were usually preceded by an epigastric aura. After two years' treatment by bromide, alone and combined with iron, and also with arsenic, she was having several fits every month. There was some improvement with bromide and belladonna, but after some months' treatment three fits still occurred each month. Digitalis, ♏v of the tincture, was then added to the same dose of bromide (℈j ter die), and she had not a single severe fit for six months, although 'sensations' continued a few times a week. She then ceased attendance, but almost immediately had another attack, and they recurred on the same treatment about one a month, and then almost ceased. During the next two years she had only one attack every four or six months, but if the medicine was discontinued the attacks immediately recurred. In this case there was a loud mitral murmur, and some dilatation of the left ventricle, the impulse being diffused, and the apex outside the nipple-line.

A man, without distinct heredity, began, at 44, to suffer from 'twitchings in the face and hands,' which continued, until at 46½ he suffered from nocturnal fits, epileptic in character, general, but more severe on the right side than on the left. Their frequency increased until, at 47, when he came under treatment, he had two a week. On bromide only they ceased for three months, and then recurred, and continued in spite of ʒss of bromide three times a day. During three months' treatment on bromide (℈j) and belladonna (♏v), three times a day, he had a fit each fortnight. The same quantity of tincture of digitalis was then substituted for the belladonna, and the fits ceased for six months, except on one occasion when he omitted the medicine for a fortnight. At the end of six months the dose was lessened, and he had some more attacks, but they again ceased when the dose was increased, and he remained free for several months, and then had two more. In this case there was distinct dilatation of the heart, and an irregular pulse.

Jesse M., aged 12, had suffered from fits since he was sixteen months old, being then backward in teething, in walking, and in talking. There was no neurotic family history. He had no doubt suffered from a considerable degree of rickets, for his legs retained traces of old curvature. The attacks occurred during both waking and sleeping states, at intervals of a week. They were preceded by a visual aura, 'a ball of fire before the eyes,' always red, and seeming to get nearer and nearer until he lost consciousness. The convulsion commenced in the right side of the face, and was more severe in the right limbs than in the left. Amm. brom. gr. xv, with tinct. belladonnæ ♏v, first twice and then three times a day, increased the interval to one month, but after six months' treatment the attacks still occurred. Tinct. digitalis, ♏v, was then substituted for the belladonna, and he had no fit for eleven months, when the dose was reduced and a fit occurred, but on again increasing the dose seven months passed, and then he had two fits, then an interval of two months, and on again substituting belladonna for the digitalis the attacks became more frequent.

A girl, aged 18, without heredity, came under treatment for fits which had occurred frequently since ten, and she had also convulsive attacks at seven and two. The interval was about two days. The fits occurred without warning, and were evidently epileptic. Bromide alone, and with belladonna, lessened a little the frequency of the attacks, but made no considerable difference, from twelve to sixteen fits occurring every month. Digitalis was then substituted for the belladonna, with the effect of reducing the fits first to four and then to two in the month, and then she passed four months without a single fit. At the end of that time they recurred, but again ceased for four months under the same treatment.

A young man, aged 22, whose father was insane, was first attacked at twenty while in the kitchen of an hotel at which he was cook, but he had had 'faintings' several times daily before the fit, and they continued afterwards, until two months later he had a second fit. After this the severe attacks occurred more frequently, four or five daily, until he came under treatment. They were preceded by a cephalic sensation, and were apparently epileptic in character. His aspect was dull, and his memory bad. Under bromide (℈j three times a day), the fits continued as before. The addition of belladonna reduced the fits, for a few months, to one or two daily, and then they increased in frequency in spite of the increase of the dose of tinct. belladonnæ to ♏xv. Tinct. digitalis, ♏v, was then substituted, the same dose of bromide being continued. During the next month he had not a single fit, and although one or two attacks occurred during the next six months, they then ceased, and he discontinued attendance after he had been free from fits for eight months, although he occasionally had a 'fainting sensation.'

The following case affords an illustration of the value of this combination in nocturnal epilepsy :—

A girl, aged 14, without heredity, had suffered from fits during sleep since eleven. The first occurred in the night after she had been frightened by a severe thunderstorm. The attacks were probably epileptic, followed by hysteroid convulsion; she was said to first jerk and then 'struggle'; the hands and fingers were at first cramped, and then 'she fought with them,' so that she had to be held down for twenty minutes.

There was no cardiac derangement. An attack occurred almost every night before treatment was commenced. Under pot. brom. ℈j, tinct. belladonnæ ♏x ter die, they were reduced to one every ten days, but did not cease, even after four months' treatment. Ammon. brom. ℈ij, tinct. digitalis ♏v, was ordered every night. During the next two months she had not a single fit, and then she ceased to attend.

In the following cases, the bromide and digitalis were given together at the first, and so their effects cannot be altogether separated. In the first, however, the influence of the variations in the dose of digitalis shows that considerable influence must be attributed to it.

A boy began to suffer from fits at fourteen, and at eighteen they occurred at intervals of seventeen days, the attacks being severe with tongue-biting. The heart's action was excited, but there was no murmur. Zinc and belladonna had no effect, but on bromide (℈j) and digitalis (♏v) the interval was increased to five and eleven weeks. A reduction of the bromide to gr. xv and the digitalis to ♏iij was followed by a return of the fits once a month, but on increasing the digitalis to ♏v, without increasing the bromide, no fit occurred, first for two and then for six months, and he has now passed another period of six months without an attack.

Adelaide B., aged 16, came under treatment January 7, 1876, on account of frequent attacks of *petit mal* during the previous two years, and occasional severe nocturnal fits for six months, in which the tongue was bitten. The latter occurred at intervals of about three weeks. Two of her father's sisters had suffered from fits. She was ordered potass. brom. gr. xv, tinct. digitalis ♏v ter die, and an aloetic pill every night. The attacks, major and minor, ceased at once. She attended for ten months, having no attack, and was heard of six months later as still perfectly well.

Henry W., aged 21¾, without inherited tendency, a somnambulist in childhood, had his first known attack nine months before being seen. He was a science student, and had been working hard at the spectroscope for some time. He had had only four attacks, all in the night without waking, and only knew of their occurrence by finding his tongue bitten and a splitting headache in the morning. He was ordered ammon. brom. ʒss, tinct. digitalis ♏ijss, every night. From the first dose to the present time, now four years, he has had no other symptom of an attack, in spite of hard work and exciting examinations. He took the medicine for fourteen months, and for the last three years has not taken more than twenty doses, keeping it by him in case he fancies he feels less well than usual.

*Belladonna*, like digitalis, rarely useful alone, is sometimes of great service in combination with bromide. It also has long been used, having been recommended by Mardorf in 1691, and at the end of the last century by Münch, Stoll, and Hufeland. I have met with no case of true epilepsy in which the attacks ceased entirely on belladonna, although, in several patients, the fits were reduced in frequency. As an adjunct to bromide it has been extensively used, and its utility is often unquestionable. It has been said to be most useful in cases of *petit mal*, but it is also effective in cases in which the attacks are severe, whether they occur by day or by night.

Its influence is probably due to its direct action on the nervous system, which is, by large doses, first stimulated and then depressed. The effect is apparently similar upon the cerebral hemispheres, the spinal cord, and the vagus and cardiac nerves. The cerebral excitement is followed by coma, the reflex action is at first increased and afterwards lessened; the heart is at first slowed and afterwards accelerated, and in consequence of the latter the blood pressure is greatly increased. The cerebral symptoms cannot be explained by the effect on the circulation, since they do not occur when a similar condition of the circulation is brought about by other causes.

The following cases illustrate the influence of the combination of belladonna and bromide in epilepsy. In the first three cases the bromide was given alone in the first instance, and the superiority of the combination was evident. In the first case the combination of belladonna and bromide had a very marked effect, although, in consequence of an increase of the dose of the bromide when the belladonna was added, the influence of the latter is not quite proved.

A young man, aged 19, suffered from fits at intervals of three weeks, usually in the morning, just before or after breakfast. They had commenced at 13 without known cause. There was no warning, and the convulsion was general and severe. Fifteen grains of bromide, night and morning, reduced the fits to one a month. After three months, a scruple of bromide and five minims of tincture of belladonna were given three times a day. No fit occurred for ten months, and then he had two. The medicine being continued, however, he had no other fits for fourteen months, and was then advised to try without medicine. He was heard of six months later, still free from fits.

Emma R., aged 35, married, without heredity, came under treatment March 1875, on account of fits which had occurred frequently since the age of 15. There was no warning, and consciousness was lost; the exact character of the convulsion could not be ascertained, but urine was often passed during the attack and it was followed by some delirious excitement with erotic tendency. Several fits occurred every week. On potass. brom. ℈j ter die, she had one fit the first month, none the second, and two the third. Tinct. belladonnæ, ♏v, was then added. She had one fit in each of the next three months, and then the attacks ceased. Four months later she had one. The treatment was continued for six months more, the dose being gradually diminished, and then she ceased to attend and remained free from fits without treatment for twelve months. A recurrence then took place, but again ceased on the same treatment.

A woman, two months after a confinement, at 43, began to suffer from fits which recurred at intervals of a fortnight when she came under treatment at 49. The fits were apparently epileptic, the warning being trembling and pain in the head. On fifteen grains of bromide and on ten grains of bromide and five of iodide three times a day the frequency of the fits remained the same. Fifteen grains of bromide with five minims of tincture of belladonna increased the interval first to three months, then to six months, and then to a year.

In the next cases the result of the combination of bromide and belladonna was very satisfactory, although they do not afford conclusive evidence of the value of the belladonna, since the treatment was commenced with the combination.

A woman, without inherited tendency, aged 29, had suffered, for a year and a half, from fits which occurred at intervals of two days to a month, always during the waking state. They were preceded by an epigastric aura, and were of moderate severity, not attended by tongue-biting. Pot. brom. ℈j, tinct. belladonnæ ♏v, were ordered twice a day. She had no other fit. At the end of nine months the dose was reduced, and at the end of twelve months the bromide was reduced to ten grains once a day. At the end of eighteen months the patient was discharged.

The following is an instance of unilateral convulsions arrested by bromide and belladonna:—

The patient was a girl, aged 18, two of whose cousins had suffered from chorea. The first attack occurred two years before, and, like all the others, commenced locally in the right arm, with flexion of the fingers and elevation of the hand to the shoulder, and affecting afterwards the whole of the right limbs. The attacks occurred frequently, and generally when she was going to sleep. There was no evidence of organic brain disease. Potass. brom. ʒss, tinct. belladonnæ ♏v, was ordered to be taken each night, and a half dose every morning. After the first dose of the medicine she never had another fit, and, eleven months later, the dose having been gradually reduced, she was discharged.

Atropine has been frequently employed instead of belladonna; one or two minims of the liq. atropiæ of the British Pharmacopœia containing $\frac{1}{120}$ and $\frac{1}{60}$ of a grain of atropine may be given. In some cases it seems to be more efficacious than the belladonna.

Eneas P., aged 16, whose father's mother was epileptic, had suffered for a year from fits, the first having occurred a few days after seeing another person in a fit. They occurred chiefly in the early morning, without warning, and were distinctly epileptic. Two or three attacks occurred each week. On bromide (℈j bis) there was no attack for twelve months; then they returned, and he had an attack each month in spite of the bromide being given three times a day with ♏v of tinct. belladonnæ. One minim of liq. atropiæ was then substituted for the belladonna, and he had only two fits in five months. The dose of liq. atropiæ was then increased to two minims, and he had no fit for eight months. Then a slight attack occurred. Nine months later, the medicine was lessened to twice a day, fourteen months' later to once a day, and after fifteen months' freedom, to every alternate day, but he then had another attack, and soon after he ceased attendance.

*Stramonium*, another solanaceous plant, was recommended in the last century by Baron Stoerck. I have had no experience of its use, but from the statements made by some old writers it deserves further trial.

*Cannabis indica*, which was first recommended in epilepsy by Dr. Reynolds,[1] is sometimes, though not very frequently, useful. It is of small value as an adjunct to bromide, but is sometimes of considerable service given separately. It may be noted that the action of Indian hemp presents many points of resemblance to that of belladonna; it is capable of causing also delirium and sleep, first depression and then acceleration of the heart, and also dilates the pupil. The cerebral excitement is relatively more marked, and the effect on the heart and pupil much less than in the case of belladonna.

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[1] *Loc. cit.* p. 321. It has been lately recommended also by Sinkler (*Philadelphia Med. Times*, Sept. 1878).

In the following case its effect was far more decided than that of bromide:—

John K., aged 40, came under treatment in 1868, having suffered from fits for twenty-five years. They occurred during both sleeping and waking, at intervals of a fortnight. There was a brief warning, vertigo, then loss of consciousness, and tonic and clonic spasm followed by some automatism;—'acts strangely and cannot dress himself.' The attacks ceased for a time on bromide, but recurred when he discontinued attendance. He came again in October 1870; scruple doses of bromide of potassium three times a day had now no effect, and the fits, at the end of four months' treatment, were as frequent as ever. Ext. cannabis indicæ gr. ⅛, three times a day, was then ordered; the fits ceased at once, 'a wonderful change' the patient declared. He had no fit for six months, and then, having discontinued attendance, the fits recurred, but were at once arrested by the same dose of Indian hemp. He continued free from fits for some months, until, during my absence, bromide was substituted for the Indian hemp: the fits immediately recurred, and he left off treatment. He returned to the hospital in six months' time, and on Indian hemp passed two months without an attack. In the third month another fit occurred, and the patient again ceased to attend, and did not return.

*Gelsemium sempervirens.*—The effect of gelsemium in lowering the reflex action in the spinal cord, and in depressing the respiratory centre and paralysing some of the cranial nerves, suggested its trial in epilepsy. The results show that its value, alone or in combination with bromide, is not considerable, although now and then it seems to be of some service, as in the following case:—

A boy, aged 14, had suffered, for seven years, from fits, each of which commenced by rotation to the left, and was succeeded by sickness, but without giddiness. Their frequency, before treatment was commenced, was not noted, but while taking bromide, bromide with digitalis and bromide with belladonna, he had from four to eight fits per month, whereas during three months in which five and seven minims of tincture of gelsemium was given with each dose (℈j) of bromide, only two fits occurred per month.

*Opium* was recommended in epilepsy by Paracelsus and by Crato and Quercetanus in the sixteenth century, and De Haen published a case in which large doses of laudanum, taken on the occurrence of the warning of a fit, cut short the attacks and cured the disease. It has, for the most part, given place to other remedies. Its alkaloid, morphia, has been advocated by Dr. Radcliffe, and I have employed it in a considerable number of cases. Now and then it is certainly effective, but much more frequently in hysteroid cases than in epilepsy. The exact nature of the attacks in the following case is a matter of some doubt, whether they were those of minor epilepsy or purely hysteroid; but morphia was more effectual than bromide in arresting them.

The patient was a little girl, aged 13, without heredity, whose first fit occurred five months before she came under treatment. She was severely frightened, one dark night, by some boys, and screamed out loudly, and all her attacks began with a similar scream. She was said to 'fight and struggle' in the attacks, but while under observation there was no convulsion or spasm. Four or five times a day, or in the night when asleep, she would scream out loudly and seem as if in a dream, but remember afterwards nothing of the scream. Bromide reduced the attacks from four or five in the twenty-four hours to one each night, but on morphia being substituted for the bromide they ceased entirely, and during four months she was kept under observation no other fit occurred.

In the 'status epilepticus,' in which attacks recur with great frequency for several days, and in which bromide often fails entirely, I have known hypodermic injections of morphia, in doses of $\frac{1}{16}$th of a grain, to be of great service, and Sieveking has found it useful, given by the mouth, in the same state. But morphia is a remedy which can only be employed hypodermically in epileptics with extreme caution. If an attack occurs, and the post-epileptic coma coincides with the sleep induced by morphia, the patient's life is in great danger. For instance, an epileptic, after a surgical operation, was injected with a quarter of a grain of morphia to relieve great pain. As the injection was beginning to take effect, he had a slight epileptic fit, and immediately passed into a condition of profound coma, with infrequent breathing, and it was necessary to maintain artificial respiration for an hour.

*Zinc* unquestionably deserves some of the repute which it has enjoyed, for more than a hundred years, as a remedy for epilepsy. Its influence is rendered intelligible by the fact that, given to animals, it has been found in large doses to lessen reflex action, and also to influence the functions of the medulla oblongata and cerebral hemispheres. It very commonly lessens the frequency of attacks, but in most of the cases in which it does so, bromide has a still more powerful effect, and zinc certainly arrests the fits far less frequently than bromide. In a few cases in which bromide fails to do good, zinc is useful, but this result was met with in only ten of the cases of this series, and in only three did the attacks cease under its use. The cases in which it was more useful than bromide varied too much to furnish ground for definite 'indications.' It is certainly useful, however, in many cases in which hysteroid convulsions succeed true epileptic fits. The oxide of zinc was the form commonly employed, in doses of three to seven grains twice or three times a day. The nausea which it causes constitutes a serious drawback to its use, as in larger doses it commonly causes vomiting, and I have never succeeded in giving the doses employed by Herpin, nor have I, any more than others, seen the remarkable effects from its use which he describes. It is best given after meals, as few patients can bear it on an empty stomach. It is probably slowly converted in the stomach into the chloride. No superior effect was obtained with the sulphate of zinc, and the bromide proved of little value, perhaps on account of the gastric irritation which any but the smallest doses produced. The following case affords an example of the beneficial influence of zinc:—

A man, aged 28, whose mother's sister was insane, had his first fit, without known cause, at 27. He had been occasionally, but not habitually, intemperate. The attacks occurred in batches of seven or eight every six weeks, always when he was awake. Each was preceded by a warning, 'dazzling before the eyes,' and consciousness of some mental disturbance. The fits were epileptic, and the tongue frequently bitten. He was ordered five grains of oxide of zinc twice a day, and attended for five months without a single attack. He then ceased attendance.

The following case illustrates the fact that zinc is capable, in some cases, of producing just the same effect as bromide :—

A girl, aged 20, whose mother's brother was insane, had her first fit at sixteen, and they had recurred, at intervals of three months, always during sleep, and evidently epileptic. Her pulse was slightly irregular, but the heart seemed healthy. Menses regular. She took ʒss of bromide of ammonium each night for six months without a fit, and then ceased attendance. She remained well for four months without treatment, and then had another fit, and a second three months later, when she returned to the hospital. Four grains of oxide of zinc were given twice a day. She took this for nine months without a single fit, and then ceased attendance.

Illustrations of the utility of zinc when the epileptic fits are followed by hysteroid convulsion were afforded by the next cases :—

A little girl, aged 8, with a history of inheritance (epilepsy in paternal grandfather and great-aunt), had had fits for a year, at intervals of a few days to two months. The warning was sudden headache, and she would fall unconscious, and sometimes bite her tongue and afterwards scream and struggle (post-epileptic hysteroid convulsion). After four months of bromide treatment she was having two fits a week. Three grains of oxide of zinc were ordered, and in two months the fits had ceased, and she continued free for six months. She then had an attack, but no other for three months more, when the last note was made.

A man, aged 26, had suffered for eleven years from fits, which occurred once a fortnight, usually in the morning; they were unattended by warning, but he would fall suddenly and then pass into a condition of hysteroid convulsion, fighting and kicking, followed by sleep for half an hour or so. But since in some attacks he bit his tongue, it is probable that in the others the hysteroid convulsion really succeeded a slight epileptic fit. His memory was bad; his pupils large. Five grains of oxide of zinc was given twice a day, and for two months he had no attack. In the next two months he had two fits, so the dose was raised to ten grains, and he had no other attack during three months longer that he remained under treatment.

Occasionally the addition of zinc to bromide distinctly increases its influence over the attacks :—

Samuel H., a dull, stupid boy, whose sister was epileptic, began to suffer from fits at eleven months 'from teething,' and they were occurring once a fortnight when he came under treatment at fourteen. The warning of the fits was giddiness, but their exact character could not be ascertained. The attacks occurred both by night and day. On bromide of ammonium (℈j three times a day), the fits ceased for several months, and the lad discontinued attendance, but returned in consequence of a recurrence. On the same treatment, and on bromide and belladonna, the attacks continued very frequent, and although they were lessened by bromide and digitalis, they did not cease until oxide of zinc was substituted for the digitalis, the dose of bromide remaining the same. The attacks then at once ceased, and when he had had no attack for eleven months, quinine was substituted for the bromide and zinc. He has now taken this for three months without any relapse.

In the following case complete arrest of the attacks was obtained by zinc, belladonna, and bromide, although bromide alone had previously been ineffectual :—

A gentleman, aged 35, whose father and brother were epileptic, had many fits in infancy, but none others till 25. Their recurrence was preceded for a short time by many fainting attacks, some of which occurred in warm rooms, &c., but others without exciting cause. He had several fits together every three months while taking bromide only, sometimes by night, sometimes by day, but always during sleep. Each was preceded by a 'swallowing sound' in the throat, and the convulsion was severe and epileptiform, lasting a few minutes only, and followed by deep sleep, and this occasionally by mental aberration for several days. His general health was good, his pulse infrequent (54). He was ordered (May 1878) to take ammon. brom. ʒss, tinct. belladonnæ ♏x, every night, and zinci oxidi gr. iij, increasing to gr. vj, every morning. Fifteen months later I heard that he had not had any attack since commencing the treatment, and although I have not heard of him since, I feel sure I should have done so had he had another fit.

I have lately employed the citrate of zinc instead of the oxide. It is rather more soluble, and is tolerated better by the stomach, and seems to be equally useful.

The addition of *arsenic* to bromide in no case produced any marked effect on the attacks. It was used in a large number of cases on account of the readiness with which, it was found, the bromide rash could be prevented by its use.

*Bromide of camphor*, which has been highly praised by Bourneville, I have tried in a considerable number of cases, but without any good result.

The combinations of *aconite* and of *hydrocyanic acid* with bromide I have also employed, but without more than very transient effect in any case.

The addition of *iodide of potassium* to bromide has been said to increase its influence. I have never been able to observe satisfactory evidence of this, having never known attacks to cease under the combination, which continued on the same dose of bromide. In the cases in which the combination was useful, there was reason to attribute the effect to the bromide rather than to the iodide. If given alone, iodide of potassium seems to have no influence on the attacks, even in doses of twenty or thirty grains, except in cases in which they are due to syphilitic brain disease.

*Iron.*—Certain distinguished authorities (as Brown-Séquard and Hughlings Jackson) have discountenanced the administration of iron to epileptics, asserting that, while it improves the health of epileptics, it increases the frequency and severity of the fits. I believe that this conclusion is only partially correct. I have given iron to several hundred epileptics, for both long and short periods, and have carefully watched its effect. Its influence certainly varies in different cases, and may be thus summarised. In rare cases it does increase the frequency of the attacks; in the majority of cases it may be taken without any ill effect on the attacks, and often with all the benefit to the general health which attends its use in other cases. In some patients its influence on the attacks is distinctly beneficial. Attacks which continue on bromide alone may cease when iron is added to it, and some patients are better on iron only than on bromide only. I have met with a few cases in which attacks which continued on bromide ceased altogether when iron was substituted.

The cases in which the attacks are distinctly aggravated by the administration of iron are so uncommon that no hesitation on this ground need ever be felt in giving iron to an epileptic in addition to the bromide or other drug which he may be taking. Of course, if, instead of adding iron, it is substituted for bromide, the effect of the cessation of the bromide must not be ascribed to the influence of the iron.

The beneficial influence of iron in epilepsy is not limited to its hæmatinic effect. It appears to do good by a specific action on the nerve-centres, similar to that which is produced by other metals, as by silver and zinc. Meyer and Williams[1] found that iron is capable of exerting such an influence. They injected tartrate of soda and iron into the blood of various animals, and found that it caused motor paralysis of central origin, the excitability of the muscles and peripheral nerves remaining intact.

Iron is of especial use in cases in which the attacks are mainly hysteroid, but it is also sometimes effective in cases which are purely epileptic, and that independently of the existence of anæmia.

The following cases illustrate some of the facts above mentioned. The first two are instances of the fact that the addition of iron does not necessarily lessen the beneficial effect of bromide treatment.

Kate P., aged 20, without indication of heredity, had fits from teething, at thirteen months, to 5. She was backward in teething and walking; did not walk until 2½. At 5 the fits ceased, to recur for a short time at 12, and again at 17, from which age they continued until she came under treatment at 20, occurring then every week, always in the waking state, without warning. They were severe epileptic attacks with tongue-biting. She had also some minor fits. Potass. brom. gr. x, tinct. fer. perchlor. ♏x, tinct. nuc. vom. ♏x, were given three times a day. The severe fits at once ceased, and the minor attacks after a few months. She continued free from fits for eighteen months, when, on omitting the medicine (the dose of which had been reduced), she had another. She has now passed another period of fifteen months without a fit, continuing the same treatment.

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[1] *Archiv für Exp. Path.* Bd. xiii. p. 70.

Marion F., whose mother was epileptic, was brought at the age of 10, on account of two epileptic fits which had occurred during the preceding fortnight. She was a delicate-looking but intelligent child. No cause for the attacks could be discovered. Ammon. brom. gr. x, ferri ammonio-cit. gr. iij, were ordered three times a day. She attended for three months, and during that time had no other attack.

The next case is an example of the fact that bromide with iron may arrest attacks on which the same dose of bromide, without iron, has little influence.

Harriet H. (whose sister was epileptic) suffered from fits in infancy, from four months to three years of age, but had no other attacks until 12½, and came under my care six months later, having then frequent attacks of *petit mal.* Small doses of bromide (ten grains three times a day) had little effect on the fits, but when ten minims of the tincture of the perchloride of iron was added, the attacks were arrested and remained absent for two months, when she ceased attendance. After discontinuing treatment the attacks recommenced, and six months later, having had some severe hysteroid convulsions (probably post-epileptic), she returned. These attacks again continued on bromide only; after several months' treatment perchloride of iron was added to the same dose of bromide: the attacks at once ceased, and after she had had no attack on this treatment for six months she again ceased attendance, and has not been heard of since.

The fact that, after bromide has arrested the attacks, iron may be given with impunity, with bromide or, after a sufficient time, instead of it, is illustrated by the next two cases.

Louisa W., aged 8, came under treatment July 1878; mental defect since birth, perhaps due to difficult labour. She was unable to talk, walked badly, and was mischievous. There was no neurotic heredity The fits commenced at five, and occurred at intervals of four or five days. There was no warning; the convulsion was general. A scruple of bromide each morning was ordered. She has now been under treatment nearly three years, and from the first dose of the medicine she has never had another fit, although for two years she has taken iron in addition to the bromide. The dose of the latter was reduced to fifteen grains after a year's treatment.

A girl, aged 17, had suffered from right-sided fits with loss of consciousness from the age of 3 years. Several seizures occurred daily. At first bromide alone was ordered: the attacks became less frequent and slighter. After three months, belladonna was added to the bromide, and the fits ceased entirely. When she had had no fits for three months, a quinine and iron mixture was substituted for the bromide, on account of general languor, and was continued for six months without any recurrence of the fits.

In the following two cases, attacks, apparently epileptoid, ceased under the administration of iron.

A married woman, aged 48, came under treatment in February, 1873, on account of 'fits' which had occurred during the preceding two years. There was a family history of fits and insanity. The attacks occurred chiefly at the catamenial periods. Only an imperfect history of their character could be obtained: she described them as being sudden attacks of inability to move or speak, lasting a few seconds, and suddenly passing away. In some it appeared that consciousness had been lost. The catamenia had not ceased; the urine was free from albumen. Tinct. ferri perchl. ♏xv three times daily was ordered. During the next two months she had a few slight attacks: they then ceased, but afterwards recurring, the dose of the perchloride was increased to ♏xx, and the fits ceased; after two months she left off the treatment, and remained free from fits for a year and a half. They then returned, also at the catamenial periods, but were arrested by bromide, and she remained free, without treatment, for twelve months. On a second recurrence she came to the hospital, and iron was again ordered, and taken for three months without a single attack occurring. She then ceased attendance.

A single man, aged 23, had his first fit five months before he came under my care. No cause could be assigned for it. There was no history of family tendency to disease of the nervous system, of sexual excess, or of syphilis. His heart and optic discs were healthy, his bowels regular. The interval between his fits was rather less than a week. They occurred usually at night, and were preceded by no warning. Consciousness was lost, and the convulsion severe, his tongue being often bitten. He was ordered tinct. ferri perchlor. ♏x three times daily. During the next month he had only one slight attack. During the following month he had no fit; during the third month he had only one fit; during the succeeding three months he had not a single attack, and he then ceased attendance. During the whole of this time he took the iron only. After ceasing to take it he remained free from fits for four months. The fits then returned, and three months later he came again to the hospital, having the fits 'as bad as ever.' He was again put on the perchloride of iron, and the fits at once ceased; he continued to attend the hospital for three months more, and during that time had not a single attack.

Iron and digitalis were of striking service in the next case. The chief feature of the convulsions was hysteroid, but they commenced with epileptiform amaurosis, and the patient had, besides, many faints very like attacks of *petit mal.*

The patient was a girl, aged 21, whose first fit occurred five months before, and was attributed to alarm at seeing a girl in an hysterical fit. Since then she had frequent fainting attacks, and slight occasional 'struggling' fits. The fainting attacks occurred several times weekly. At the commencement of each fit her sight would be lost, 'everything becoming black, without a glimmer of light,' and then she 'struggled,' being held down with difficulty. Her heart was healthy. Tincture of steel (♏x) and tincture of digitalis (♏iij) were ordered. A slight faint occurred a few days after the treatment commenced, but no other symptom of attack for four months, at the end of which time the patient ceased attendance.

*Borax.*—In several cases of epilepsy in which bromide had no effect, I have given borax with considerable benefit. It is said to have a sedative effect in large doses. In many of the cases in which I have given it, little effect was produced on the disease, but in some its influence was decided, and greater than that of any other remedy. The dose varies from fifteen grains to half a drachm, three times a day. It is not well to begin with a larger dose than fifteen grains, because, in some patients, it causes at first some diarrhœa, occasionally with dysenteric evacuations. This, however, which is not very common, disappears if the dose is reduced, and does not recur when the dose is subsequently increased. It may be taken for a long time without inconvenience, but in three cases an eruption of psoriasis occurred—in one patient after it had been taken daily for two years, and in the second (communicated to me by Dr. Spencer of Clifton) after nine months' administration, and in the third after it had been taken for eight months. In each case, however, the eruption quickly disappeared when arsenic was added to the borax.

Robert C., without heredity, began to suffer at 16 from fits, preceded by loss of speech. He came under treatment at the age of 32, but did not pass under my care until he was 37, when he was having the attacks frequently under bromide treatment, two having occurred during the preceding ten days. Before a fit, for three or four minutes, he was unable to speak or to write, although he could go on with his work. Then his head became confused, and he fell unconscious, with a variable amount of epileptoid convulsion. In February 1878, gr. xv of borax was ordered twice a day, and he had not a single fit until September, when he had another, and during the next twelve months he had a very slight fit every two or three months. In September 1880, he omitted the medicine, and had two attacks. Since then he has taken borax continuously for two years, the daily dose having been gradually increased to ʒjss during the last four months. He has had no ill effect from it except slight diarrhœa at the commencement, and although he still has an occasional attack, he always says that borax has done him more good than any medicine he took previously.

The influence of borax over fits is very well shown in the following case, and it is interesting because bromide was more effectual after the borax treatment than before, and it shows clearly that if borax does good for a time, and then seems to lose its power, bromide should again be tried, even though it was at first useless.

Emma B., single, aged 14, without heredity, had suffered from fits, evidently epileptic in character, for fifteen months. The first occurred soon after being awakened out of sleep by a fire near. The attacks occurred during the waking state, at intervals of two weeks. They were without warning, and severe. Bromide alone was first given for two months, but the attacks were rather worse than better, several occurring each month. On belladonna alone the weekly average was 1 and 1·5. Ten grains of borax three times a day was then given, and the fits at once ceased, and she had not an attack for five months. The dose was then ordered to be taken only twice a day, and she had a fit almost immediately. The third dose was resumed; a fit occurred the following month, and then four months passed without an attack. A fit occurred in each of the two following months, so bromide (℈j three times a day) was substituted, and up to the present time (fourteen months) she has had no other fit.

My experience of combinations of borax with other drugs has not been large, but some cases I have met with show that these are occasionally of service. In the following obstinate case, for instance, the effect of the combination with gelsemium was very striking:—

Arthur O., without heredity, suffered from fits in infancy, between one and two years of age; dentition and walking were both late. The attacks continued from that time until he came under treatment at 17, in January 1877. The interval varied from one to six days. The fits were sudden, without warning, slight, followed by some mental aberration (automatism) for two hours. During two months' treatment on bromide the attacks were uninfluenced. On five grains of oxide of zinc three times a day he had two fits a month, but only one fit a month on seven, ten, and twelve grains. Tinct. ferri perchlor. was then substituted; during the first month he had one fit; during the next month none, but afterwards the attacks became more frequent, and on other treatment (the notes of which were lost) the attacks became very numerous, so that in one week he had fifty-four. Ten grains of borax three times a day were then given, and for the first week he had no fit; then some attacks in the night, but none in the day; the latter recurred after some weeks, and the dose was increased to twelve grains, and five minims of tinct. gelsemii added. The fits at once stopped, and in January 1881 he had not had a single fit for sixteen months, although the dose had been reduced in June. The medicine was then discontinued, and he has not been heard of since.

*Mistletoe.*—This old remedy for epilepsy, praised by Paracelsus and by a considerable number of writers since his time, especially by Colbatch (1723) and Fraser (1806), I have tried in a considerable number of cases, but with beneficial result only in a case in which the attacks were apparently hysteroid—the patient whose seizures have been described on p. 169.

*Turpentine* was originally recommended by Pritchard and Foville,[1] and has been praised by Watson and Radcliffe. I have, however, seen no good result from its administration in epilepsy, although in hysteroid attacks it is unquestionably useful.

*Cocculus Indicus.*—The use of cocculus indicus in epilepsy, introduced by Dujardin-Beaumetz, has lately attracted attention in consequence of the recommendation of Planat. I have tried the alkaloid picrotoxine in a few instances, but in only one case has it appeared to do good. My own experience of its use has, however, been small, and I am very much indebted to my colleague, Dr. Ramskill, for permitting me to mention some interesting results which he has obtained by the hypodermic injection of picrotoxine. His experience of its effect on the fits when given through the skin is nearly the same as my own of its employment by the mouth. In seven cases in which it was injected, in daily doses of from one to four milligrammes, no beneficial result was obtained; in most cases, indeed, the attacks were rather more frequent and severe. Of course, we are not justified in assuming that the effect of picrotoxine and of the cocculus indicus itself are identical. A very interesting fact has, however, been ascertained by Dr. Ramskill—viz., that picrotoxine in larger doses of from fifteen to eighteen milligrammes will almost invariably produce a fit in twenty or thirty minutes. In one patient, for instance (according to the notes of Mr. Broster, who carried out the experiments), the dose was daily increased, and when more than five milligrammes were injected a sensation of giddiness followed, similar to that with which the attacks commenced. The same effect followed larger injections, and when the dose reached eighteen milligrammes a severe attack occurred in thirty minutes, and an attack always followed the injection of this dose. In another patient a similar progressive increase of the dose was followed by giddiness and headache when eight milligrammes were injected. When the dose of fifteen milligrammes was reached, a severe epileptic fit followed. Next day a second dose of fifteen milligrammes did not cause a fit; but eighteen milligrammes, two days later, caused a fit in half an hour. After a week's intermission, twenty-four milligrammes were injected, and a severe fit occurred in twenty-five minutes. In a third patient a fit occurred after one injection of eight milligrammes, but ten milligrammes next day caused no fit. Fifteen milligrammes, however, were followed by a fit in thirty minutes; and a second injection of the same dose the following day caused a fit in fifteen minutes. Seventeen milligrammes next day caused a fit in thirty minutes. In a fourth patient a single dose of eighteen milligrammes caused, in ten minutes, giddiness and slight dazzling before the eyes, and in thirty minutes there occurred the usual aura of an attack—a sensation of something creeping up the right arm to the top of the head, and numbness and twitching in the right thigh; but no fit followed, although the patient was stupid and dull for a time just as after a fit.

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[1] *Dict. de Méd. et de Chir. Prat.*, 1831, art. 'Epilepsie.'

The one solitary exception to the otherwise uniform failure of cocculus indicus, in the instances in which I have tried it, is the following case:—

A girl, aged 16, began to suffer from the attacks, without known cause, a year before she came under treatment. She had only severe fits, which occurred once a month, always in the early morning. Each was preceded by a sensation in the hypogastric region, felt on waking, and this sensation 'worked up to the heart,' and then she lost consciousness. The fit was epileptic, lasting two or three minutes. Five minims of tincture of cocculus indicus three times a day were ordered. During the first month she had no fit, during the second one only, during the third one, but that occurred after she had omitted the medicine for a few days. The dose was then increased to ten minims. During the next four months she had not a single attack, and she then ceased attendance (October 1880). I have lately learned that in January 1881 she had a fit, but has had no others up to the present time (May 1881).

Chloral hydrate I have not found useful in ordinary cases of epilepsy, alone or in combination with bromide. From nitrate of silver and sulphate of copper, remedies which in the past enjoyed a high repute, I have seen, in the few cases in which I have tried them, no resulting benefit. Among other drugs which I have tried and found useless are benzoate of soda, nitro-glycerine, Piscidia Erythrina, codeia, Calabar bean, ergot, sclerotic acid, and nitrite of amyl given by the stomach.

*Counter-irritation*, by applications to the skin of the scalp or neck, has been frequently employed, the method which is most frequently adopted being a seton in the neck. That it occasionally does good appears to be undeniable, and an extensive accidental burn sometimes produces a similar effect. In most cases the relief has, however, been temporary only, and it frequently fails. In the few cases in which I have tried it, not the slightest effect on the attacks could be observed.

*Trephining*, a very old remedy for epilepsy, has lately been again brought into prominence in consequence of the more exact localisation of diseases of the brain, which furnishes the surgeon with a more accurate indication of the locality for the operation. It is justifiable only in cases in which the disease has followed an injury to the head, and in which either there is still depressed bone, or the local commencement of the fits suggests that the disease causing them is at the surface of the brain, involving the motor convolutions which are adjacent to the fissure of Rolando. I have met with only one case of epilepsy which fulfilled these conditions, and in that the patient had been trephined for the original fracture, and the fits dated from an attack of hemiplegia which came on six months after the accident, and was apparently due to a secondary lesion, probably thrombosis. In no other case of epilepsy after an injury to the head have the convulsions been local in their commencement. But although it is, in my opinion, justifiable only in such cases, it is right to state that cases have been reported which were benefited by the operation, although no localising symptoms existed, and even in which, strange to say, no condition was revealed by the operation which explained its influence.[1] It is possible that the measure is sometimes beneficial as an energetic form of counter-irritation; but it is doubtful whether, for this object, its performance is justifiable.

*Castration* has been proposed as a remedial measure, and has been performed without effect. It has been lately revived by Bacon[2] as a means of arresting epilepsy due to masturbation in adult insane patients. In boys, however, *circumcision*, if effectually performed, is usually successful, and should be adopted in all cases in which there is reason to associate the disease with masturbation.

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[1] For an extensive collection of facts bearing on the subject, the reader is referred to a paper by Echeverria in the *Arch. Gén. de Méd.* 1878, pp. 529 and 652. Interesting cases of benefit from trephining have been lately recorded by Agnew (*Philad. Med. Times*, 1878, p. 578), West (*Med. Chir. Trans.* 1880), and Lees and Bellamy (*Clin. Soc. Trans.* 1881).

[2] *Journal of Mental Science*, Oct., 1880, p. 470.

### ARREST OF INDIVIDUAL ATTACKS.

An important detail of the treatment of many cases of epilepsy is the arrest of commencing attacks. It is of course only in the case of fits which are ushered in by a deliberate warning that the question of their arrest presents itself. The means by which attacks may be arrested have been already described (p. 93). The attempt is most frequently successful in cases of convulsions beginning unilaterally in the hand or the foot, in which a ligature around the limb, or the forcible extension of the contracting muscles, is sometimes effective. These methods should always be tried in such cases. The most effective way of employing the ligature is for the patient to fasten a piece of tape round the arm so that it can readily be tightened. If the tape is doubled, passed round the limb, with the ends through the loop formed by the doubled part, the extremities may be brought down to the lower part of the sleeve, so as to be accessible and readily pulled tight, as soon as the warning is felt.

The method is occasionally very efficacious, and is not only effective in the arrest of individual attacks, but also as a means of producing a more permanent effect. I have already (p. 95) mentioned the case of a patient whose fits, after having been many times arrested by the ligature, stopped spontaneously at the spot at which the ligature had previously been applied. A similar case was recorded in the last century by Lysons.[1]

It has been proposed by Brown-Séquard and by Buzzard to produce a more permanent effect, in the cases in which the ligature is effective, by applying a circular blister around the limb.[2] In some cases this method is effective. In one patient, described by Buzzard,[1] it led to a transfer of the commencement of the fits from one arm to the other, and some years later, the patient having died of phthisis, I found that there existed a small tumour in the white substance above one lateral ventricle.

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[1] *Pract. Essays upon Intermitting Fevers*, 1772.

[2] The method is not entirely modern. Alexander Trallianus, who lived soon after Galen, describes a case in which the attacks commenced by a sensation beginning on the back of the foot and ascending to the head. The fits were arrested by creating an issue on the part from which the attacks seemed to start. The treatment by local blisters was employed also by physicians in the seventeenth and eighteenth centuries.

It has been seen (p. 94) that there is reason to ascribe the effect of the ligature to an increase in the resistance in the part of the brain to which the discharge is spreading. The peripheral sensory impression, acting on the cells of the sensory centre, seems to raise their resistance, or that of the connected cells of the motor centre, so as to arrest the discharge, and the permanent arrest of fits must be ascribed to a corresponding change in the 'functional nutrition' of the centre. The effect of the blister appears to be to cause a similar change more suddenly. This sudden alteration, in some cases, by opposing suddenly a considerable resistance to the discharge, appears to divert it into other paths, and I have met with cases in which the arrest of attacks by the ligature, and still more by the blister, was attended by so much giddiness and other distressing feelings, that the patients declared that the arrested fit was worse than the full attack. Such cases are, however, not frequent.

I have tried other means of increasing the central resistance by peripheral impressions, especially by daily faradisation of the limb with a wire brush, and by circular sinapisms, but hitherto without success. It seems as though the more profound the alteration in the peripheral nutrition, the greater is the effect on the function of the nerve-centre.

Attacks which begin by other warnings are sometimes arrested by other forms of peripheral sensory impression, by a strong gustatory sensation, as by chewing a piece of ginger, by a strong olfactory impression, as by the application of ammonia to the nostrils, or by swallowing a handful of common salt (Nothnagel). More uniformly successful, however, is the inhalation of nitrite of amyl.

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[1] *Practitioner*, Oct. 1868.

If it produces flushing of the face before the patient loses consciousness, the attack is usually cut short. The cases in which I have found it most frequently successful have been those with a deliberate olfactory aura. Why this should be is difficult to explain, since it is scarcely conceivable that the effect of amyl is produced through the olfactory nerve. Its influence may be held to afford some support to the doctrine that the sudden contraction of the cerebral vessels is the essential element in the attack; but it is intelligible that a sudden increase in the blood supply to the brain should change the form of cerebral action, whatever be its original cause, and also that the drug may act directly upon the nerve-tissues, just as it does apparently upon the nerve-cells of the vasomotor system. The patient whose deliberate warning is described on p. 67 would not, on any account, be without his bottle of amyl, and it was equally useful in the case of another patient with an olfactory warning. An incident which once occurred to this patient may be mentioned as illustrating a possible inconvenience of the drug. One day, when out, he felt an attack coming on, and smelt the amyl, but too late to arrest the fit. He fell unconscious, spilling the contents of the bottle in his fall. A person a few yards away, who saw him apply the bottle to his mouth and at once fall down insensible, rushed to him, and, seeing the empty bottle labelled 'Poison,' sought the nearest policeman, and asked him to go to a man who had just committed suicide. When the policeman arrived at the spot, he found, to his surprise, the supposed suicide preparing to walk away. Such an accident may be prevented by the use of the glass capsules of nitrite of amyl now to be obtained.

The inhalation of chloroform sometimes arrests an attack, but it is rarely that the opportunity to try this agent presents itself, except during the status epilepticus. In this it is occasionally useful, although too often the attacks recur when the effect of the chloroform passes off.

Injection of apomorphia has been recommended as a means of arresting epileptic fits by Vallender.[1] I have had no experience of its employment in these attacks, although I have long used it for cutting short hysteroid seizures.

### TREATMENT DURING AN ATTACK.

Little treatment is needed during the attacks of epilepsy. In patients who bite the tongue, a cork, or better, a small piece of indiarubber, should be forced between the teeth, and thus the tongue-biting may often be prevented. The patient should be laid down for obvious reasons. The horizontal posture has been recommended on account of the theory of vascular spasm and cerebral anæmia; but I am doubtful, from observation, whether the horizontal posture has much influence on either the duration or the severity of the attack.

It is necessary to see that the clothes are loose about the neck. If tight, when the neck becomes turgid and swollen, the resistance to the return of blood from the head is increased, and extravasations into the skin or conjunctivæ are more probable.

Little can be done to arrest the developed attack. Only in the early stage is an attempt to cut it short by any method successful. The hysteroid condition, however, which succeeds a slight fit, may often be cut short by the means presently to be described. The sleep which in many cases succeeds a fit should not be prevented. There is often more headache if the patient is kept awake than if permitted to sleep. The administration of a small quantity of alcohol after an attack does no harm, but it is rarely needed or beneficial. The weakness and prostration pass away in a little time without its aid.

In the status epilepticus, described on p. 193, bromide often fails. Inhalations of nitrite of amyl have been found useful by Crichton Browne. Chloroform inhalations rarely have a permanent effect. The remedies from which

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[1] *Berlin. Klin. Wochenschrift*, 1877, p. 185.

I have seen most good are repeated doses of chloral (gr. xv. every three or four hours), the subcutaneous injection of morphia (in doses of gr. ½ or ⅛), and the application of ice to the spine.

### GENERAL MANAGEMENT OF EPILEPTICS.

*Diet.*—It has been recommended, on theoretical grounds, that the diet of epileptics should contain little or no animal food. In a few observations which I have made by keeping a patient under unaltered medicinal treatment for alternate periods on a diet with and without animal food, I could observe no difference in the attacks, except that in one patient they were slightly more frequent in the periods when animal food was excluded, and in another attacks which were apparently hysteroid, on ordinary diet, became, when meat was excluded, severe epileptic fits, and again became hysteroid when animal food was restored.

Stimulants should be taken sparingly by epileptics, in small quantities, as a glass of wine with a meal. Alcohol in such amount is not prejudicial, but any actual stimulation, sufficient to be followed by a reaction, should be prohibited. In young persons who have not been accustomed to stimulants, I think that alcohol is far better avoided altogether.

In all cases it is most desirable that regular action of the bowels should be secured. Nothing conduces to the occurrence of attacks more than constipation and gastric disturbance.

*Occupation.*—A moderate amount of exercise, physical and mental, is undoubtedly desirable for epileptics, but severe and exhausting exertion of any kind is undesirable. There is no necessity for the education of children to be arrested. A moderate amount of brain-work will do good rather than harm. The excitement of competitive and other examinations is, however, undesirable. But it must be remembered that there are many positions in life for which the liability to epilepsy constitutes no insuperable disqualification, and for which it is desirable that the unfortunate sufferers should be, as far as possible, prepared.

In the choice of a permanent occupation so much depends upon personal opportunities that it is difficult to lay down general rules. But there is one consideration to which all others must be subordinate—the occupation must not be one which involves any risk of life in the event of the occurrence of attacks. Hence all work among machinery, all work which involves the ascent of ladders, or a seafaring life, must be prohibited. An outdoor life is rather better than sedentary occupations, but the choice of the latter is so much the larger that in most cases a sedentary calling has to be selected.

*Marriage.*[1]—The question of marriage presents itself in two aspects, as regards the individual and as regards the possible offspring. As regards the disease, marriage in itself has little influence. The ancient opinion that continence may cause, or sexual intercourse cure, epilepsy, is probably now held by none, and is certainly as opposed to facts as it is disastrous in its practical effects. Attacks which have resisted treatment before marriage usually persist afterwards without any considerable change. There is no evidence to show that moderate sexual intercourse has any influence on the disease, except in rare instances of the first coïtus or in which the act habitually induces an attack. It certainly has never a beneficial effect, but it is, as a rule, prejudicial only when excessive or exhausting. Nor is the disease in women materially influenced by either the pregnant or the puerperal states. A few patients have more frequent attacks during pregnancy; a few have complete immunity during the period; in the majority the attacks are uninfluenced by the condition.

As regards the offspring, however, the question of marriage is very different. Although the children frequently escape, there can be no doubt that they are far more liable than other children to this or other allied neuroses, to suffer from infantile convulsions, or to be epileptic or insane, or idiotic.[1]

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[1] An interesting account of the medical, popular, and legal opinions on the relation of marriage and epilepsy has been lately given by Echeverria: 'Marriage and Hereditariness of Epileptics,' *Journal of Mental Science*, October 1880.

From the facts stated on p. 23, and from the statistics of Echeverria quoted below, it is clear that the risk of fatal infantile convulsions is far higher than in other children, and it is probable that one child in seven, perhaps more, will be epileptic. The risk of transmission exists even if the disease is acquired, but is far less than when the disease in the parents is inherited, being three times as great in the latter case as in the former, the proportions, according to Echeverria, being one in five, and one in fourteen. Regarding the probability that the children will suffer from other neurotic diseases, these figures, for the reason stated in the note, scarcely afford the ground for an opinion.

The high probability that some of the offspring will suffer from epilepsy or other nervous diseases should be clearly stated to those who seek an opinion on the propriety of marriage. There is no certainty that the taint will be transmitted. On the contrary, as regards any individual child, the probability is in favour of its escape, and if the disease in the parents is acquired, the likelihood of escape is considerable. But the probability is also against the escape of all the offspring from disease of the nervous system, and when the disease in the parents is inherited, the probability is very great that if there are many children, some will suffer, and that by a still larger number, the disease will be handed on to other generations. In the interests of the individual, of the family and of the race, it is therefore desirable to discourage as far as possible the marriage of epileptics.[1]

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[1] On this point the only statistical evidence are some facts which have been recently published by Echeverria (*loc. cit.* 364). He investigated the health of the children (553 in number) of 136 married epileptics, and found that no less than 195 (35 per cent.) died in infancy of convulsions; 78 (14 per cent.) were epileptic; 18 (3 per cent.) were idiots; 11 (2 per cent.) insane; and 39 (7·5 per cent.) were paralysed, and only 105 (19 per cent.) were perfectly free from nervous troubles. It is probable that these figures somewhat exaggerate the effect of the transmitted taint. Infantile convulsions are largely due to other causes, and are, among the children of the poor, extremely common apart from inherited tendency. Moreover, the proportion of healthy children is lowered by the exclusion of children who are 'choreic' or 'hysterical,' *i.e.* who suffer from morbid states which, while doubtless dependent in part on the transmitted taint, are of slight importance in regard to the practical question. On the other hand, the majority of the children had not reached adult life, and many were yet young, and hence, probably, the number of cases of insanity is far, and that of epilepsy considerably, below the actual facts; and as the ages of the children are not stated, the amount of probable error from this cause cannot be conjectured.

### *HYSTEROID OR COORDINATED CONVULSIONS.*

The treatment of the attacks which present hysteroid or coordinated convulsion varies according to their character and relation. This relation has been seen to be complex. Cases in which the chief visible phenomena of the attacks is hysteroid may be primarily epileptic or primarily hysteroid, or may be so far intermediate between the two diseases that it is difficult to know in which category to place them. The treatment must vary according to the essential nature of the attacks. When the disease is primarily epileptic, and the hysteroid phenomena are merely 'post-epileptic' disturbances, the treatment has to be, in the main, that for epilepsy, and bromides commonly do good. If, on the other hand, the attacks are primarily hysteroid, bromides usually fail, while other remedies often succeed.

Of these one of the most important is iron, which is frequently beneficial, as in epilepsy, apart from the existence of actual anæmia. The form of iron does not appear to be of importance. I have seen the greatest benefit from the tincture of the perchloride, but the ammonio-citrate, the citrate of iron and quinine, and the sulphate of iron, in the form of the aloes and iron pill, are all useful. It is important to guard against the constipating effect of iron by the administration of an aperient. In some cases the iron alone is effectual, in others it is better to combine it with bromide.

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[1] Some curious facts have been collected by Echeverria on the measures to which the recognition of this fact has at times given rise. He cites the following quaint passage from the *Croniklis of Scotland,* by Hector Boethius, translated by John Bellenden, Edinburgh, 1536: 'He that was troublit with the fallin evil, or fallin daft or wod, or havand sic infirmite as succedis be heritage fra the fader to the son, was geldit, that his infectit blude suld spread na firther. The women that was fallin lipper, or had any infestion of blude, was banist fra the company of men, and gif she consavit barne under sic infirmity, baith she and her barne were buryit quik.'

The following cases are examples of the utility of iron in these cases:—

A pale delicate-looking lad, aged 12 years, with a strongly phthisical family history, had his first fit nine weeks before he came under my care. The attacks had recurred frequently; he had had four during the week before he was first seen. Their character (according to the account carefully and repeatedly obtained) was peculiar. Each was preceded for a few minutes by an aching in the left eye, and it was said that the eye sometimes became red: he then fell unconscious, the face being not pale, but flushed; there was first violent 'trembling' of his limbs, and then co-ordinated movements in which he would sometimes struggle, and clutch and try to bite, and sometimes put his head between his legs and remain rolled up in a ball (see p. 178). His left pupil was a little larger than the right; the optic discs were full-tinted, certainly redder than the average, but within the range of health. Tinct. ferri perchloridi ♏x was ordered three times a day. In the following two weeks he had only one fit each week instead of four. The medicine was then omitted for a week, and he was brought to the hospital with the statement that he had had a fit every day whilst he was not taking the medicine. During the following month the medicine was taken rather irregularly, and the boy's mother asserted that the fits ceased when it was being taken, and as soon as it was omitted they recurred. During the following month it was taken more regularly, and the boy had only two fits. There was no alteration in the ophthalmoscopic appearances. The dose was then increased to ♏xv. During the first fortnight after the increase there were no attacks; during the next, apparently from some accidental cause, there were five. The iron was, however, persevered with, and for five months he had not a single fit. He remained pale and delicate in appearance, and on the medicine being discontinued he had some more attacks, which ceased when the iron was resumed.

Eliza T. came under treatment at 18, her first fit having occurred four months before, five minutes after a fright, and the second the following day. They had recurred daily, sometimes as frequently as twenty a day, always when awake. In one of her attacks which was seen, the eyes were turned up, and she fell gently to the left; the hands were clenched, the legs rigid, with occasional internal strabismus, and opisthotonos, the whole ceasing suddenly. On aloes and iron pill, with valerianate of zinc, the attacks were reduced to one or two a month. On bromide and valerian she had several attacks daily. On oxide of zinc the attacks were much less frequent, but still occurred. On perchloride of iron, ♏x three times a day, they became gradually less frequent, and on increasing the dose to ♏xv they ceased, and after she had had no attack for two months, she discontinued attendance.

In the case of another patient with violent hysteroid attacks in which iron was distinctly more useful than any other remedy, the effect of treatment is shown in the following table:—

| Treatment | Weeks | No. of fits | Weekly average |
|---|---|---|---|
| Zinci brom. gr. v ter . . . . | 3 | 13 | 4⅓ |
| Potass. brom. ℈j ter . . . . | 1 | 9 | 9 |
| „ c. tinct. bel. ♏x . . | 2 | 5 | 2½ |
| „ „ ♏xv . . | 1 | 2 | 2 |
| „ „ ♏xx . . | 1 | 3 | 3 |
| „ „ ♏xxv . . | 4 | 7 | 1¾ |
| Mist. terebinth. . . . . . | 1 | 4 | 4 |
| Tinct. ferri perchlor. ♏xx ter . . | 5 | 2 | ⅖ |

Tincture of valerian is an old remedy for these attacks, and is sometimes useful, but it rarely arrests them altogether. Valerianate of zinc is more often efficacious. Other forms of zinc are not often of service. Morphia is occasionally distinctly useful. Turpentine is a very old remedy for hysteria, and is certainly far more useful in such cases than in ordinary epilepsy. In the case of the lad whose severe attacks are described on p. 165, their arrest was apparently due to this remedy. In the little girl whose prolonged hysteroid attacks and mental disturbance are described on p. 156, turpentine was the only remedy which was of distinct service. It should be given in ten minim doses emulsified by mucilage or by rubbing up with pulv. acaciæ, and diluted with peppermint or camphor water. It is well to push the dose until slight symptoms of strangury appear.

The utility of turpentine in these cases is intelligible from modern experiments on its action which have proved that its first effect is to lower the cerebral functions, leaving reflex action unaffected until a comparatively late period. This renders also intelligible its inutility in ordinary epilepsy, in which the drugs most serviceable are those which effect a primary reduction in reflex action. The depression of the cerebral functions is in harmony with Pritchard's opinion that the drug has a peculiar sedative and tranquillising influence on the nervous system, although it appears that the effect is produced in a manner different from that by which bromide produces the same result.

The following case also illustrates the utility of turpentine in this form of attack :—

A lad, admitted at the age of 13, had suffered from frequent fits for eighteen months, the first having occurred a week after being stunned by a blow on the head. The attacks began suddenly without warning, and consisted of violent co-ordinated movements which commenced at the first moment of the attack. While sitting at dinner, for instance, he suddenly threw his arms about, knocked a glass of beer over, kicked and plunged, and talked wildly about a patient with whom he had had an argument. The attack lasted for five minutes, and ended suddenly, and after it he immediately sat down and finished his dinner. In some attacks he got his tongue between his teeth, but it was never much bitten. His mind was natural. The effect of treatment was as follows :—

| | Days | No. of fits | Weekly average |
|---|---|---|---|
| Pot. brom. gr. x, liq. arsen. ♏iij ter . . | 18 | 5 | 2 |
| „ gr. xv „ „ . . | 7 | 4 | 4 |
| Amm. brom. gr. xv ter . . . . . | 28 | 22 | 5 |
| Pot. brom. gr. xx, mag. sulph. ʒss ter . . | 27 | 7 | 2 |
| Ol. terebinth. ♏xv ter . . . . . | 52 | 0 | 0 |

*Treatment during Hysteroid Attacks.*—In pure epilepsy, as already stated, the only treatment needed during the attacks is such care as shall secure the patient, as far as possible, from injury. It is very different with the attacks of hystero-epilepsy, which, from their character, severity, and long duration, often furnish the attendants with a task of no small difficulty, and which can, almost always, be cut short by appropriate treatment. The patients often hurt themselves during the attacks, and some control is absolutely necessary. But, as already stated, restraint tends to increase the violence and makes the paroxysm last longer. Hence considerable judgment is often required, so to adjust control as to be efficient and not too much. I have seen these patients put within padded partitions and left alone, but I have never myself found this necessary.

The slighter attacks can be arrested by closing the mouth and nose with a towel for some twenty or thirty seconds, after Dr. Hare's method. The profound effect on the respiratory centre, and the related higher centres, caused by the anoxæmia, seems to arrest the convulsive action. Cold water over the head is often successful if applied freely; in severe attacks a moderate quantity only excites redoubled violence, while a second gallon is often more effectual than the first. This has the disadvantage of drenching the patient's head, and often gives cold. When the mouth is open during the attacks a small quantity of water poured into it is often successful. A much more convenient and more effectual remedy than water, however, is strong faradisation to the skin; applied almost anywhere it will commonly quickly stop the attack. Of ovarian compression I have already spoken (p. 159). In this country it is rare that ovarian pressure will arrest an attack. In some cases all these means fail, even when thoroughly used; and I have known such attacks go on, in spite of skilled treatment, for several hours. Chloroform is of little use; its administration is a matter of extreme difficulty, often impossibility, and the attack is commonly renewed when the influence of the anæsthetic passes off. The remarkable effect of nausea in relaxing spasm led me some years ago to try the effect of injections of apomorphia, and I have found in it an unfailing means of arresting the attacks. After the injection of a twelfth of a grain, in four minutes, with certainty, all spasm ceases, and normal consciousness is restored; in six minutes the patient will sit up; in eight minutes will vomit; and afterwards, except for slight nausea, is well. A twentieth of a grain has the same action, but is rather longer in its operation. Moreover, I have found that the treatment is often, so far as the hysteroid symptoms are concerned, curative as well as palliative, for the attacks in many cases ceased after a few paroxysms had been thus cut short.

A good illustration of the effect of apomorphia in arresting these attacks is afforded by the following notes of one patient, kept by the Resident Medical Officer, Mr. Broster:—

The patient, Mabel S., 25 years of age, had suffered from attacks, at intervals of two or three days, for a year. They occurred by both day and night. 'The patient goes quietly off on to the floor; there is internal strabismus, and rapid opening and closing of the eyelids. The limbs are stiff and rigid, and the body is rotated to one side or the other. Then follows violent struggling, with noisy breathing, gasping, &c., the face being flushed. This lasts three or four minutes, and the patient then passes into a quiet drowsy condition, and if roused at once goes off into the same series, and this succession of attacks will last for half an hour, an hour, or an hour and a half, and afterwards she is much exhausted. She never bites her tongue, but is extremely dangerous to the attendants, as she bites their hands and arms.' Soon after the commencement of one of these attacks, $\frac{1}{12}$ grain of apomorphia was injected. Within five minutes she became paler, came to herself, got up and walked to the lavatory, vomited freely, lay down on the bed and went to sleep, and woke up well. She had no fit for four days, but on the fifth day she had another fit. Immediately after the commencement $\frac{1}{16}$ grain of apomorphia was injected. The hysteroid convulsion was very violent at the time, but in five minutes it had entirely ceased, and five minutes later she vomited freely. During the next three weeks the patient had only one attack in the day, although she had several during the night. During the fortnight before her discharge she had only one slight attack of any kind.

Harriet B., aged 23, came under treatment in February 1878 for attacks which had occurred almost daily for six months. Her sister had suffered from chorea, but there was no other neurotic family history. The commencement was attributed to overwork. In the attacks she fell down and lay still and stiff for a minute or two, and then began struggling and kicking, and was usually brought round by water being thrown on her. On April 2, during an attack, $\frac{1}{12}$ grain of apomorphia was injected. In three minutes later the attack had ceased, and in seven minutes she vomited. Another attack was cut short on April 4 in three and a half minutes. After this there were no more fits.

The treatment of the cases of this class has, however, frequently to be other than by drugs. The occurrence of the primarily hysteroid attacks is largely influenced by the moral state of the patient. I do not mean that they are deliberately brought on, or that their actual occurrence is under the control of the patient, but it is certain that the mental state in which they occur is to a large extent influenced both by external circumstances and also by the patient's will. Defective volition, imperfect mental control, is a characteristic of these patients. Under annoyance the patient 'works' herself or himself into a mental and moral state in which the occurrence of an attack is inevitable. The attack itself often cannot be prevented, but the mental state may be prevented to a large extent.

The greatest advantage to the patient results from a complete change of 'moral atmosphere' by removal from home influences, which are often unfavourable by reason of annoyances, or of the unwise solicitude of friends, which fosters instead of controlling the disease. The mere admission into a hospital is often sufficient of itself to arrest the attacks altogether. A large part of the benefit, however, depends upon the character of the person under whose immediate care and control the patient is. Some nurses, by their tact and firmness, are notoriously more successful in dealing with such cases than others. Strangers often succeed where relatives, however wise, have, by the mere fact of their relationship, little influence.

Other tonic measures are also useful, especially the shower bath and the needle bath. In the latter jets of water play upon all parts of the body from a series of circular pipes.

Where symptoms of derangement of the uterine functions exist, it is of course important that this should be put right. Recorded cases, in which the attacks have ceased when a uterine displacement was rectified, have not been paralleled by any facts which have come under my personal observation.

The management of many of these cases of chronic convulsive disease is a task of difficulty, requiring the utmost patience and perseverance on the part of both the patient and the physician. The old power of casting them out has gone from the earth, and it is only by the study of their origin and history, and careful experiment in their treatment, that we can hope to regain over them such power as may still be possible to man. And the present generation has witnessed an advance in the treatment of these diseases equalled in perhaps no other branch of therapeutics. Thanks to the influence of one drug and its combinations, hundreds of epileptics have been cured, and thousands are leading useful lives who would otherwise have been incapacitated by the disease. Although the condition of many sufferers is still gloomy enough, it is not without hope, and to them also, we may surely trust, the progress of the recent past is the dawn of a brighter day.

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LONDON : PRINTED BY
SPOTTISWOODE AND CO., NEW-STREET SQUARE
AND PARLIAMENT STREET

Catalogue B]

*London, New Burlington Street*
*May*, 1881

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